



**ATLAS OF**

**CARDIOVASCULAR DISEASES**



# ATLAS OF CARDIOVASCULAR DISEASES

CORRELATION OF CLINICAL ELECTROCARDIOGRAPHY  
AND CARDIAC ROENTGENOLOGY WITH  
CLINICAL HISTORY AND AUTOPSY FINDINGS

By

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To  
MY WIFE

CLARA M KARR TREIGER M D

Whose help and encouragement have been a great stimulation  
In tribute to her constant inspiration  
this book is affectionately dedicated



## PREFACE

A good clinical history is of paramount importance for a correct cardiac diagnosis. The analysis of clinical symptoms and signs found on physical examination must be made in terms not only of abnormal anatomy, but of physiology as well. After all it is the disturbance of the normal physiology, frequently the sequence of abnormal anatomy, which brings the patient to the clinician.

Röntgenological examination of the heart provides the clinician with data as to the gross anatomy of the heart with changes in size, shape and position of the heart and its various chambers. The measurement of size is of little value; it is of far greater importance to find out which chambers are involved in abnormal process. To obtain complete information the left and right oblique views must be taken in addition to the anterior, as the greater posterior portion of the left ventricle and also both auricles are situated posteriorly and can be visualized only in oblique views.

Electrocardiography provides the clinician with valuable information as to the physiology of the heart especially the myocardial conduction. It records any disturbance in initiation or conduction of the impulse and any deviation in the electrical axis. By correlating with clinical data the clinician interprets the electrocardiographic changes in conduction in terms of abnormal anatomy and physiology as well.

Correlation of history, physical findings, roentgenogram and electrocardiogram leads to a correct understanding of the degree of anatomical and physiological changes in the heart and to a proper dose of various medications for their correction.

Regardless of how convincing one method of examination may be, the interpretation of it without correlation with other findings may lead to misdiagnosis and failure of therapy.



Frequent examinations and comparative studies of roentgenograms and electrocardiograms are essential in deciding whether there is any activity of the abnormal anatomical or physiological changes found

This atlas is divided into six parts: Normal Heart, Rheumatic Disease, Arteriosclerotic Heart Disease, Hypertension, Syphilitic Heart Disease, and Congenital Anomalies

A cross section of common types of heart disease with autopsy findings is presented. In selection of the cases many difficulties were met, especially as to availability of autopsy, roentgenographic and electrocardiographic findings in the same case. An endeavor has been made to present various cases with characteristic changes shown in the roentgenogram, electrocardiogram, pathological specimen, emphasizing their **clinical** values. Only simple, uncomplicated cases are presented. By visualizing the elementary changes in uncomplicated cases of cardiac disease, it is much easier to analyze the complicated anatomical and physiological changes of some cardiac disease with more than one etiological factor.

I wish to express my thanks and grateful appreciation to Dr. William G. Hibbs, Medical Director, and to the members of the Medical Department of the Presbyterian Hospital for kind permission to use some of their cases.

IRVING I. TRIFIGER

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## NORMAL HEART

For a clinical evaluation of abnormal anatomical and physiological changes in the heart, a knowledge of normal anatomy, as shown by the roentgenogram and physiology, especially myocardial conduction as shown by the electrocardiogram, is essential.

The anterior view of a roentgenogram demonstrates the aorta, pulmonary artery, and the smaller anterior portion of the left ventricle, the outflow tract. Both auricles and the greater portion of the left ventricle, the inflow tract, are located posteriorly and visualized in the left and right oblique views.

In a normally built adult, the heart is in an oblique position while in a tall thin person it is vertical and in a stout, short person horizontal. In young children the heart is globular and centrally located. The electrocardiogram shows variations which are normal for that particular type of heart.

A normal heart is rotated by scoliosis of the spine. In scoliosis with convexity of the spine to the right, the heart is rotated counterclockwise, and in the anterior view, the heart contour is similar to that of the right oblique view. In scoliosis with convexity of the spine to the left the heart is rotated clockwise, and in the anterior view the heart contour is similar to that of the left oblique view.

A normal heart may be displaced to the right or to the left as by pneumothorax, fluid or adhesions. Except for a shifting axis, the electrocardiogram is normal in a rotated or displaced heart.

The electrocardiogram of a normal heart shows a normal initiation and transmission of the electrical impulse, a normal myocardial conduction as evidenced by normal waves (P, QRS, T) and intervals (PR, QRS).

The average weight of a normal heart in an adult is 250 (female) to 300 (male) grams. The thickness of the myocardium of the left ventricle is 12 to 14 millimeters and that of the right is 3 to 4 millimeters.

## PLATE 1 NORMAL HEART—ANTERIOR VIEW

**Anterior Position**—The patient's sternum is in contact with the film cassette or fluoroscopic screen. The patient is facing the cassette in roentgenography or the examiner in fluoroscopy. The x-ray tube is back of the patient and therefore this position is called also postero anterior (PA).

**Roentgenogram, Anterior View**—In a normally built adult the heart is in oblique position.

### Left cardiac border

- 1 Convex, upper portion—aortic knob
- 2 Concave, middle portion—pulmonary artery. The pulmonary conus of the right ventricle is medially to the right and the hilum to the left. The pulmonary conus is a cone-like portion of the right ventricle situated immediately below the pulmonary valve (see Plate 64). The hilum contains secondary branches of the pulmonary artery (see Plate 56).
- 3 Straight lower portion—left ventricle anterior part is the outflow tract of the left ventricle with blood flowing out from the apex to the aorta (see Plate 15). The greater portion of the left ventricle and both auricles are located posteriorly and are visualized in the left and right oblique views (see Plates 2 and 3).

### Right cardiac border

- 1 Upper portion—ascending aorta
- 2 Lower portion—right auricular appendage. The body of the right auricle is located posteriorly.

### Anterior surface of the heart

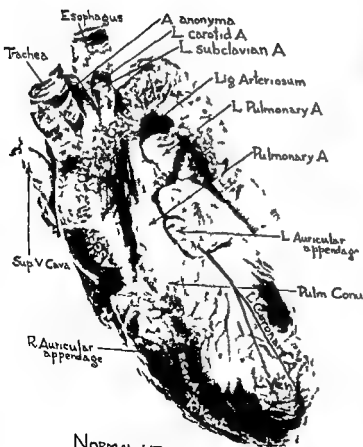
The greater central portion is the right ventricle (RV) anterior part with the pulmonary conus above and diaphragmatic surface below. The wide right lateral part is the right auricular appendage while the narrow left lateral part is the anterior portion of the left ventricle.

**Anatomy**—Correlate the anatomy and the roentgenogram. Compare with Plate 15 anterior view. The small portion of the heart below the right auricular appendage is the diaphragmatic surface of the right ventricle and is not visible on the roentgenogram.

**Summary**—The anterior view of a roentgenogram demonstrates the aorta, pulmonary artery and the smaller anterior portion of the left ventricle, the outflow tract. Both auricles and the greater portion of the left ventricle, the inflow tract are located posteriorly and not visualized in this view.

In fluoroscopy while the left ventricle pulsates inward the pulmonary artery pulsates outward. The point of opposite pulsations is the upper end of the interventricular septum and the left ventricular border.

# PLATE 1 NORMAL HEART—ANTERIOR VIEW



NORMAL HEART ANTERIOR VIEW



The patient is facing the cassette or the fluoroscope screen



## PLATE 2 NORMAL HEART—LEFT OBLIQUE VIEW

**Left Oblique (LO) Position**—The patient's left shoulder is turned toward the fluoroscopic screen or film cassette. The patient (heart) is rotated clockwise around the longitudinal axis about 50 degrees. To obtain a correct position, bisect the 90 degree angle, formed by the anterior and lateral positions, thus placing the patient at 45 degrees. Rotate the patient then slightly 5 degrees more, in the same direction. Move the tube 2 feet closer to the patient, thus taking the oblique views at a distance of 4 feet.

**Roentgenogram, Left Oblique View**—The spine is on the left side. The clockwise rotation brings the left auricle and the posterior part of the left ventricle to the front.

**Left (posterior or spinal) cardiac border**

- 1 Upper portion—left auricle. The left bronchus and aortic arch are over the triangularly shaped, periled left auricle. The border of the left auricle adjacent to the left bronchus is a straight line.
- 2 Lower portion—left ventricle posterior part, inflow tract with blood flowing in from the left auricle to the apex. The normal left ventricle barely touches the spine, and the border is slightly convex.

The heart in the left oblique view is pear shaped.

The angle formed by the lower end of the left ventricular border and the diaphragm is the interventricular groove and the lower end of the interventricular septum. The upper end of the septum is the point of opposite pulsations in anterior view (see Plate 1, description).

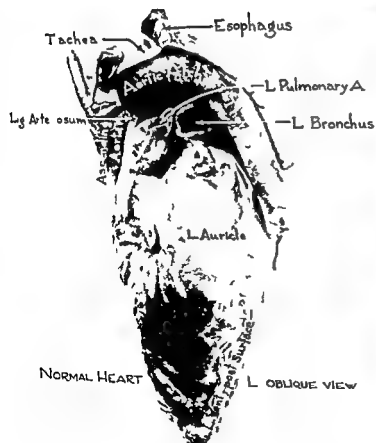
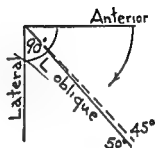
**Right (anterior or sternal) cardiac border**

- 1 Upper portion—ascending aorta.
- 2 Middle portion—right auricular appendage.
- 3 Lower portion—right ventricle anterior part.

**Anatomy**—The heart is rotated clockwise around the longitudinal axis. Correlate the anatomy and the roentgenogram. Note the left auricle with the left bronchus and aortic arch above and the left ventricle posterior surface, below. Compare also with Plate 15 and note the outflow and inflow tracts of the left ventricle. The tip of the clockwise rotated heart lies on the diaphragm and is not visible on the roentgenogram.

**Summary**—The left oblique view demonstrates the arch of the aorta, the left auricle and its relation to the left bronchus and the posterior, the greater portion of the left ventricle, the inflow tract. The normal heart in the left oblique view is pear shaped.

# PLATE 2 NORMAL HEART—LEFT OBLIQUE VIEW



The patient's left shoulder is turned toward film cassette or fluoroscopic screen

## PLATE 3 NORMAL HEART—RIGHT OBLIQUE VIEW

**Right Oblique (RO) Position**—The patient's right shoulder is turned toward the fluoroscopic screen or film cassette. The patient (heart) is rotated counterclockwise around the longitudinal axis about 50 degrees. To obtain a correct position bisect the 90 degree angle formed by the anterior and lateral positions, thus placing the patient at 45 degrees. Rotate the patient then slightly 5 degrees more, in the same direction. The x-ray tube is at a distance of 4 feet from the patient.

**Roentgenogram Right Oblique View**—The spine is on the right side. The counterclockwise rotation brings both auricles to the front on the right border while the aortic knob, pulmonary artery and the left ventricle on the left disappear posteriorly.

The heart in the right oblique view is triangular in shape with its base on the right side.

Right (posterior or spinal) cardiac border

- 1 Upper portion—left auricle
- 2 Lower portion—right auricle. The left auricle anatomically situated above the right, the septum lying horizontally between them.

Note the translucent retrocardiac space with the esophagus in it.

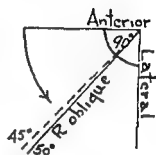
Left (anterior or sternal) cardiac border

- 1 Upper portion—ascending aorta. Note the chimneylike appearance.
- 2 Middle portion—pulmonary conus of the right ventricle.
- 3 Lower portion—right ventricle anterior part with border close to the anterior chest wall.

**Anatomy**—The heart is rotated counterclockwise around the longitudinal axis. Correlate the anatomy and the roentgenogram. Note the close proximity of the tip of the left auricle to the straight esophagus. Note the ascending aorta, pulmonary conus of the right ventricle and the anterior portion of the right ventricle on the left cardiac border. Compare also with Plate 5 and note the relationship of the left auricle to the esophagus. The tip of the counterclockwise rotated heart lies on the diaphragm and is not visible on the roentgenogram.

**Summary**—The right oblique view demonstrates the left and right auricles and their relation to the esophagus. Due to counterclockwise rotation the aortic knob, the pulmonary artery and the left ventricle on the left border disappear posteriorly and are replaced by the chimneylike aorta, the pulmonary conus of the right ventricle and the anterior portion of the right ventricle with border close to the anterior chest wall. The normal heart in the right oblique view is triangularly shaped. While taking the right oblique view give patientarium to visualize the esophagus and its relation to the left auricle.

# PLATE 3 NORMAL HEART—RIGHT OBLIQUE VIEW



NORMAL HEART  
R OBLIQUE VIEW



The patient's right shoulder is turned toward the film cassette on fluoroscopic screen

## PLATE 4 NORMAL VARIATIONS IN POSITION OF THE HEART

In a normally built adult the heart is in an oblique position see Plate 1

**Fig A Vertical Heart**—It appears long and narrow, the diaphragm is low, as seen in tall, slim persons

G F male white, aged 41 Height 5 feet, 9 inches weight 121 pounds  
Functional soft systolic murmur heard in the mitral area

In marked cases the heart appears suspended and drop shaped (drop heart) In vertical heart the contour of the pulmonary artery may form the larger part of the contour below the aortic knob The anatomical apex is directed medially

**Electrocardiogram** : QRS<sub>1</sub> low voltage

**Fig B Horizontal Heart**—The diaphragm is high as seen in short obese persons or when the diaphragm is pushed upward by a pregnant uterus large ovarian cyst ascites etc The anatomical apex is directed laterally The left ventricular border remains straight thus differentiating it from left ventricular hypertrophy or dilatation

**Electrocardiogram** : QRS<sub>1</sub> of low voltage or low notched inverted with T<sub>1</sub> frequently inverted also

**Fig C Globular Heart**—Child aged 3½ female white The heart is centrally located and its borders reach about equally far to the left and right Normal in children only

**Electrocardiogram** : QRS<sub>1</sub> inverted right axis deviation is frequently seen in children

**Summary**—In a normally built adult the heart is in an oblique position while in a tall thin person it is vertical and in a stout short person horizontal In young children the heart is globular and centrally located

The electrocardiogram shows shifting in electrical axis which is normal for that particular type of heart In vertical heart the change in QRS is low voltage is in Lead I while in horizontal heart it is in Lead 3 frequently associated with inverted T<sub>1</sub> Inverted QRS<sub>1</sub> is often found normally in children especially with globular hearts

# PLATE 4 NORMAL VARIATIONS IN POSITION OF THE HEART



Fig A Vertical heart The diaphragm is low



Vertical heart QRS: low voltage

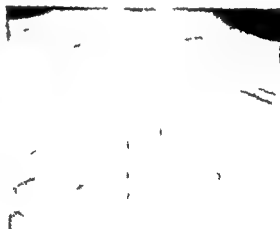
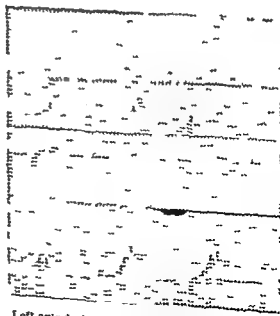


Fig C Colular heart (child)



Left axis deviation QRS: diphasic QRS: inverted

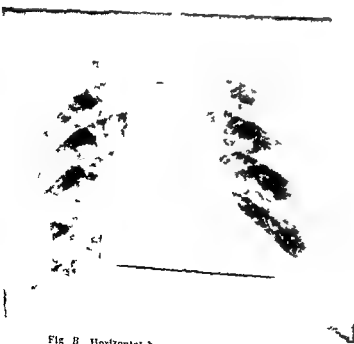


Fig B Horizontal heart The diaphragm is high

## PLATE 5 ROTATED AND DISPLACED NORMAL HEARTS

**Fig A**—Scoliosis with convexity of the spine to the right

The heart is rotated counterclockwise around the longitudinal axis as in the right oblique position. Compare with the roentgenogram Plate 3.

Note the disappearance of the aortic knob and apparent straightening of the left upper cardiac contour due to rotation. The left lower cardiac border is formed not by the left ventricle but by the anterior portion of the right ventricle. Note also the translucent retrocardiac space. The heart is triangular in shape.

**Fig B**—Scoliosis with convexity of the spine to the left

The heart is rotated clockwise around the longitudinal axis as in the left oblique position. The round shadow on the left is that of the breast. Compare with the roentgenogram Plate 2. The heart is pear shaped.

**Fig C**—The heart is displaced to the right—acquired dextrocardia

*I T female white aged 26. Far advanced tuberculous process in the right lung pulled the heart completely into the right side of the chest. Note gas in the stomach below the left diaphragm, the stomach being in normal position thus ruling out situs inversus.*

A normal electrocardiogram in this case ruled out a congenital dextrocardia which shows characteristic electrocardiographic changes (see Plate 62).

**Summary**—Scoliosis rotates the heart and changes its contour in the roentgenogram. In the anterior view, the heart contour is similar to that in the left or the right oblique view. A marked degree of rotation of the heart in scoliosis may cause twisting of large blood vessels with circulatory disturbance and cardiac failure in the absence of cardiac disease.

A normal heart may be displaced to the right and simulate a congenital dextrocardia which may be ruled out by a normal electrocardiogram.

# PLATE 5 ROTATED AND DISPLACED NORMAL HEARTS

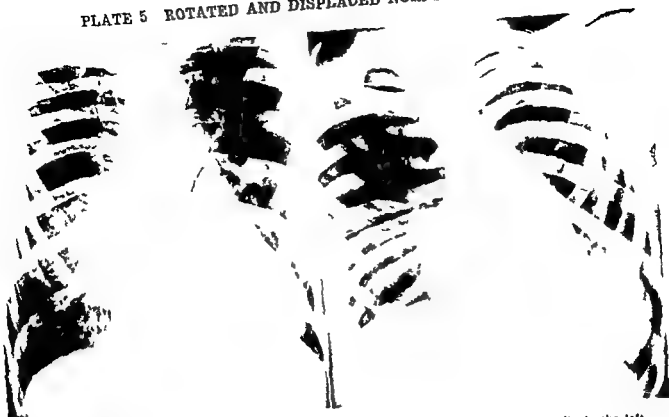
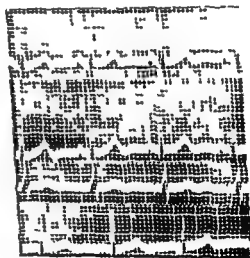


Fig. A Scoliosis with spine convexity to the right. The heart is rotated counterclockwise as in right oblique position.

Fig. B Scoliosis with spine convexity to the left. The heart is rotated clockwise as in left oblique position.



Fig. C Acquired dextrocardia. The heart is displaced completely to the right and obliterated by the density of the right chest.



Acquired dextrocardia normal ECG. In congenital dextrocardia the heart is a mirror picture and shows characteristic ECG changes. Leads I and aVR completely inverted. Leads II and III replace each other. (See Plate 6.)



## PLATE 6 NORMAL ELECTROCARDIOGRAM

The electrocardiographic record is produced by the effect of the action current of the heart which is led off from various parts of the body

Lead 1 Right arm to left arm RA to LA

Lead 2 Right arm to left leg RA to LL

Lead 3 Left arm to left leg LA to LL

Lead 4 Precordium to left leg LL wire to precordium LA wire to LL

The precordial, chest foot (CF) lead is placed on the intersection of the midclavicular line and fifth interspace so called Position 4 on the chest (CF<sub>4</sub> lead) Recently the use of two additional precordial leads in other positions on the chest was adopted as routine with a total number of six precordial leads in special cases. The three or more precordial leads give additional information as to the activity in the ventricular myocardium, anterior portion

The horizontal and vertical lines on the electrocardiogram are for measurements of rate PR and QRS intervals and voltage of P QRS and T. The horizontal measurements are for time. One small square is equivalent to 0.04 second, while one large square is equivalent to 0.2 second. The vertical measurements are for voltage (height or depth). One small square is equivalent to 0.1 millivolt and is 1 millimeter in height.

The rate is normally 60 to 100 beats per minute. The usual length of the mounted electrocardiographic record each lead is slightly over 6 inches or 30 large squares or 6 seconds in time. The number of cycles (the space between two QRS complexes) in a 6 inch strip multiplied by ten gives the approximate heart rate.

The PR interval is the conduction time from the sinus node to the ventricles. The upper limit of normal in adults is 0.20 second, in children it is 0.16 second.

The QRS interval is the intraventricular conduction time and is measured at the base of the QRS. The upper limit of normal in adults is 0.10 second in children it is 0.09 second.

The normal rhythm is the so called sinus rhythm with normal position and contour of P in reference to the QRS.

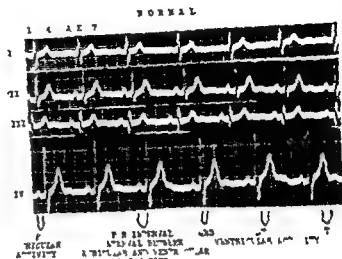
A normal electrical axis in the electrocardiogram is shown by an upright QRS in Leads 1, 2, 3.

**Summary** The electrocardiogram gives us valuable information as to the physiology of the heart: the rate, rhythm, conduction in the myocardium of the auricles and ventricles and conduction systems. This information correlated with clinical and roentgenologic findings is of great value as to estimating the anatomical changes in the heart if any are present.

# PLATE 6 THE CONDUCTION SYSTEM OF THE HEART AND NORMAL ELECTROCARDIOGRAM



AV Conduction System



LEAD 1 R CH 70 LAD 7 2 ALAD 3 SA → LA  
2 RIGHT LAD 7 3 LAD 7 4 SA → LA  
3 LAD 7 4 LAD 7 5 LAD 7 6 LAD 7  
4 LAD 7 5 LAD 7 6 LAD 7 7 LAD 7

Sinoatrial (SA) and atrioventricular (AV) conduction systems

A plexiform network of neuromuscular tissue spread through the walls of the heart. Sinoatrial node situated in the wall of the right atrium near the orifice of superior vena cava. Atrioventricular node situated in the septal wall of the right atrium immediately above the opening of coronary sinus.

The impulse starts at the sinoatrial node (the pacemaker) and is transmitted to the myocardium of both atria. P deflection in ECG is due to atricular activity. From the atrium the impulse is transmitted to the atrioventricular node. From here the conduction fibers as the bundle of His run forward on the septum.

PR interval in ECG (measured from the beginning of the P to the beginning of the QRS) indicates the time required for the transmission of the impulse from the sinoatrial node to the ventricles.

The His bundle separates into right and left branches to spread out over the ventricles.

QRS and T deflections in ECG are due to ventricular activity.

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Lead 1 Right arm to left arm RA to LA

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The horizontal and vertical lines on the electrocardiogram are for measurements of rate, PR and QRS intervals and voltage of P QRS and T. The horizontal measurements are for time. One small square is equivalent to 0.04 second while one large square is equivalent to 0.2 second. The vertical measurements are for voltage (height or depth). One small square is equivalent to 0.1 millivolt and is 1 millimeter in height.

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The normal rhythm is the so called sinus rhythm with normal position and contour of P in reference to the QRS.

A normal electrical axis in the electrocardiogram is shown by an upright QRS in Leads 1, 2, 3.

**Summary** The electrocardiogram gives us valuable information as to the physiology of the heart: the rate, rhythm, conduction in the myocardium of the auricles and ventricles and conduction systems. This information correlated with clinical and roentgenologic findings is of great value as to estimating the anatomical changes in the heart if any are present.

trophy sets in early, as well as physiological adjustment of the blood pressures. The more pronounced is the regurgitation, the more there is hypertrophy of the left ventricle as seen on the roentgenogram. In myocarditis and pericarditis, electrocardiographic changes may completely disappear, or if the scar tissue and fibrosis interfere with conduction permanent electrocardiographic changes may remain as residue of a healed condition. At this stage a murmur as evidence of a valvular lesion, auricular or ventricular hypertrophy, electrocardiographic and roentgenographic changes are signs of an inactive, healed and well compensated process, and the patient does not need any treatment for his condition.

If for any reason a breakdown of compensation occurs, a cardiac failure with failure of the function of the ventricular myocardium sets in and the failing heart must be helped by the physician to carry on the required physiological processes or a complete breakdown will lead to death.

At this stage of breakdown of compensation the physician must evaluate the extent of anatomical and physiological impairments in the heart. He must correlate the electrocardiographic and roentgenographic findings, as, for instance, the extent of auricular or ventricular hypertrophy with clinical signs of left or right ventricular failures. The knowledge of the degree of cardiac damage and its adjustment by the heart is most helpful to the physician as to the proper dose of the medication to be used.

This introduction to rheumatic heart disease would be incomplete if we failed to mention bacterial endocarditis as a complication of a healed process, of which more will be said in a special chapter assigned to it.

## 1 MITRAL STENOSIS

The function of a normal mitral valve is (1) to close during the ventricular systole in order to prevent the blood from flowing back from the left ventricle into the left auricle, the closure of the valve causes the normal first mitral heart sound which is therefore systolic in time. (2) to open during the ventricular diastole in order to let the blood flow from the left auricle into the left ventricle.

## II

### RHEUMATIC HEART DISEASE

In dealing with rheumatic heart disease the examining physician must ascertain first whether the condition is active or inactive. The terms acute, subacute, and chronic, as used by the pathologist when handling pathologic specimens, should not be used by clinicians dealing with human beings with various normal and abnormal active and inactive, physiological processes. Fever, leucocytosis and a high sedimentation rate are examples of some of the general signs of activity, while the appearance of a murmur, pericardial rub, or electrocardiographic changes are some of the topical, local signs of activity in the heart.

Active rheumatic fever may affect the endocardium with special predilection for valvulitis with mitral or aortic insufficiency or both and left arrhythmias.

The myocardium may be affected primarily or secondarily, by extension from the endocardium. The inflammation may involve the ventricular wall or the interventricular septum or both.

The pericardium, the same as the myocardium, may be affected primarily or secondarily from extension from the endocardium and myocardium, causing pericarditis or pericarditis. Pericardial rub, roentgenograms and electrocardiographic changes as low voltage, inversion of all the T waves, are some of the important diagnostic signs. The electrocardiographic abnormalities if present are valuable clues in doubtful or unsuspected cases of pericarditis.

The stage of activity and healing is followed by an inactive stage of a healed process with anatomical and physiological adjustments. Following mitral insufficiency a fibrosis of the valve sets in causing a mitral stenosis which is indication of a healed condition, with moderate or marked hypertrophy of the left auricle to compensate for it. The more pronounced is the stenosis the more there is hypertrophy and enlargement of the left auricle as shown on the roentgenogram. In aortic insufficiency the aortic valve has considerably less tendency to fibrosis and stenosis than the mitral valve. Left ventricular hyper-

deviation. The extent of the left auricular enlargement and cardiac rotation caused by it, and shown on the roentgenogram is in direct proportion to the degree of mitral stenosis.

When fibrosis and stenosis of the mitral valve is associated with auriculitis, this may cause abnormal auricular myocardial conduction, which will manifest itself in the electrocardiogram by deformed P wave, notched high or low, diphasic or inverted. The anatomical changes in the left auricle may cause increased irritability and formation of auricular premature systoles, paroxysmal auricular tachycardia (which is a succession of numerous auricular premature systoles), or, as frequently is the case, auricular fibrillation with abnormal initiation and conduction of auricular impulses.

Auricular fibrillation *per se* is not a sign of left or right ventricular failure. It is only an abnormal auricular rhythm and may be associated with a ventricular rate, which is slow, normal or fast. The fast rate unless it is paroxysmal, is evidence of ventricular failure and must be treated accordingly with digitalis. Clinically, there may be dyspnea with or without edema and cyanosis, while the electrocardiogram may show low voltage of QRS and P's as evidence of myocardial insufficiency. While auricular fibrillation in mitral stenosis may be frequently a precursor of cardiac failure there are a great number of cases in which it may persist for many years without any evidence of failure.

As long as the mitral stenosis is well adjusted or compensated by hypertrophy and dilation of the left auricle and hypertrophy of the right ventricle, the mitral stenosis, if uncomplicated is an anatomical residue of an old healed rheumatic heart disease, well adjusted and therefore is such does not require any treatment. When a breakdown of compensatory mechanisms occurs, cardiac failure with impaired function of the right ventricle develops as cyanosis, peripheral edema, ascitic enlargement of the liver, hydrothorax, complicated by thromboembolic processes which may cause the patient's death if the events cannot be controlled in time.

The anatomical changes in mitral valvulitis manifest themselves clinically by appearance of a murmur. In the active stage, the mitral valve is impaired to such a degree that it cannot close sufficiently during the ventricular systole, and blood will regurgitate into the left auricle, the murmur formed will be systolic in time, occurring during ventricular systole. In the inactive stage the fibrosis and mitral stenosis will interfere with opening of the valve and normal blood flow during the ventricular diastole, and therefore the murmur will be diastolic in time. This diastolic murmur depends in its formation on auricular systole (P wave in the electrocardiogram), which occurs in the late phase of ventricular diastole and shortly preceding the ventricular systole (QRS, ST, T in the electrocardiogram), the murmur in mitral stenosis is therefore late diastolic or so called presystolic. When mitral stenosis is complicated by auricular fibrillation, the normal mechanism of auricular systole (P wave in the electrocardiogram) is gone, and therefore the murmur cannot be presystolic any more, it becomes a mid diastolic murmur, occupying the mid diastolic phase of diastole. The presystolic mitral murmur is normally a rumble of the same intensity. The rumble is usually heard in the localized mitral area, is not transmitted, and may be influenced by a change in the patient's position and by exercise.

As the apex is displaced to the left and down on account of the rotation of the heart, the rumble is usually heard in the sixth inter-space near the anterior axillary line. The blood, while flowing from the left auricle into the left ventricle, meets with a considerable resistance of the stenotic valve, therefore the diastolic murmur is of rumble quality. This is in contradistinction to a soft diastolic murmur of aortic insufficiency when blood regurgitates freely from the aorta into the left ventricle and does not meet any resistance in the valve during its flow.

Anatomical changes in the mitral valve fibrosis and stenosis, are soon adjusted or compensated by hypertrophy and dilatation of the left auricle and later by hypertrophy of the right ventricle. The enlargement of the left auricle progresses gradually posteriorly toward the esophagus compressing it, and to the right and anteriorly, thus rotating the heart counterclockwise on its vertical axis. These findings can be well shown on the roentgenogram and in some cases the electrocardiogram, by high peaked P's especially P and right axis

# **PLATE 7 MITRAL STENOSIS WITH ADVANCED CARDIAC FAILURE**



Heart in situ Heart rotated counter clockwise by the enlarged left auricle

Mitral valve as seen from above Marked mitral stenosis Marked dilatation and hypertrophy of the left auricle

The right auricle and auricular appendage are enlarged due to thrombosis and cardiac failure and pushed out on the right side by the hypertrophied and enlarged right ventricle

Due to rotation the pulmonary cone of the right ventricle bulges on the left upper border the hypertrophied anterior portion of the right ventricle forms the left lower cardiac border

Due to counterclockwise rotation the aortic knob pulmonary artery and anterior portion of the left ventricle with the apex are displaced posteriorly

The left auricle is markedly distended The iliac is gray and opaque The mitral valve is markedly altered Instead of the usual opening there is a linear slit having a maximum diameter of about 12 to 15 mm and when stretched it is 2 to 4 mm wide The two leaflets are adherent at their adjoining margins to produce a complete singular leaflet the anterior leaflet has a maximum width of 2.8 cm the posterior 1 cm where the two leaflets join posteriorly the auricular surface is jagged and the crevice is filled with reddish brown material resembling clotted blood



## PLATE 7 MITRAL STENOSIS WITH ADVANCED CARDIAC FAILURE

J. C., male, aged 54. Chorea in childhood. In the past ten years a gradually progressive dyspnea developed, recently so severe that he needed three or four pillows at night. The symptoms progressed until the patient was weak and emaciated, and apparently in the last stage of cardiac failure.

The physical examination revealed a pale, dehydrated, thin male, acutely ill, sitting up in bed and breathing with difficulty. Pulse 92, temperature 98, respirations, 24. Heart: the apex beat diffuse and distant; a diastolic murmur heard at the apex, heart action irregular due to auricular fibrillation. Lungs: slight hydrothorax. The liver palpable five fingerbreadths below the right costal border, the edge sharp, ascites with pitting edema of both feet. Moderate amount of albumin in the urine. Two days after admission he was having difficulty with speech and was disoriented. Three days later he was having involuntary micturition and was comatose, cyanotic and had increased respirations with gradual failure of the pulse. He died six days after admission.

**Autopsy**—Chronic infective mitral aortic and tricuspid endocarditis, mitral stenosis, marked dilatation and hypertrophy of the left auricle and right ventricle, dilatation of the tricuspid orifice, thrombosis of the right auricular appendage.

Generalized passive hyperemia, chronic passive hyperemia of the lungs, slight sanguinolymphohydrothorax, thromboembolism of the branches of the right pulmonary artery, slight icterus.

Mixed sclerosis of the pulmonary artery, thoracic and abdominal aorta, coronary arteries patent.

**Summary**—A case of mitral stenosis with advanced cardiac failure is presented to demonstrate the heart in situ and the stenotic mitral valve.

# PLATE 7 MITRAL STENOSIS WITH ADVANCED CARDIAC FAILURE



Heart in situ Heart rotated counter clockwise by the enlarged left auricle

Mitral valve as seen from above Marked mitral stenosis Marked dilatation and hypertrophy of the left auricle

The right auricle and auricular appendage are enlarged due to thrombosis and cardiac failure and pushed out on the right side by the hypertrophied and enlarged right ventricle

Due to rotation the pulmonary conus of the right ventricle bulges on the left upper border the hypertrophied anterior portion of the right ventricle forms the left lower cardiac border

Due to counter-clockwise rotation the aortic knob pulmonary artery and anterior portion of the left ventricle with the apex are displaced posteriorly

The left auricle is markedly distended The lining is gray and opaque The mitral valve is markedly altered Instead of the usual opening there is a linear slit having a maximum diameter of about 1 to 1.5 mm and when stretched it is 2 to 4 mm wide The two leaflets are adherent at their adjoining margins to produce a complete singular leaflet the anterior leaflet has a maximum width of 1.5 cm the posterior 1 cm where the two leaflets join posteriorly the auricular surface is jagged and the crevice is filled with reddish brown material resembling clotted blood

## PLATE 8 MITRAL STENOSIS

E F, female, white, aged 47 died at home in June, 1944

Longitudinal section of the heart, rotated counterclockwise on its vertical axis by an enlarged left auricle

The stenosis of the mitral valve is of marked degree so that it is difficult to pass the little finger through it. This stenosis is characterized by thickening of the leaflets, obliteration of the commissures, thickening of the chordae tendineae and retraction especially of the posterior leaflet all characteristics of chronic indurative mitral endocarditis such as is observed in rheumatic heart disease. The wall of the left auricle is 3 to 5 millimeters thick and dense from proliferation of gray fibrous tissue. It is increased in size from dilatation and its lumen is partially filled with masses of mottled tan and brownish tan thrombi resembling those of a mixed thrombus.

The longitudinal section of the heart shows that the left auricular enlargement extends posteriorly toward the esophagus, compressing it and to the right and anteriorly, thus rotating the heart counterclockwise on its vertical axis. The enlargement extends to the left only as far as the left auricular appendage, and therefore cannot be visualized in the roentgenogram along the left cardiac border.

**Summary**—A case of mitral stenosis is presented to demonstrate the close similarity of cardiac outlines in the roentgenogram and in the heart specimen. The longitudinal section demonstrates the extent of the left auricular enlargement posteriorly compressing the esophagus and to the right and anteriorly rotating the heart counterclockwise on its vertical axis.

# PLATE 8 MITRAL STENOSIS



A

B

A Heart rotated counterclockwise as in right oblique position. The aortic knob is hardly visible, the pulmonary conus of the right ventricle is below it, and the anterior part of the right ventricle forms the lower portion of the left cardiac border. The apex is rounded and displaced to left and down.

B Note close similarity of cardiac outlines here and in roentgenogram.



Esophagus  
Distended with mitral

Left auricular appendix

Mitral stenosis

Longitudinal section of the counterclockwise rotated heart as in right oblique position. Esophagus is indented by an enlarged left auricle.

## PLATE 3 INACTIVE MITRAL STENOSIS

L. M., female white, aged 32

Acute rheumatic fever at 15 years of age three months in bed. Well and active since, except for slight dyspnea on exertion. A presystolic rumble heard in mitral area not transmitted. Blood pressure, 100/70. No physical findings of aortic or other valve involvement. Roentgenograms show characteristic changes due to enlarged left auricle.

**Anterior View**—Compare with normal, Plate 1

The heart has slightly rotated counterclockwise on its vertical axis and therefore the left cardiac border has partially disappeared posteriorly. The aortic knob is hardly visible. The pulmonary artery contour, which is normally concave, has due to rotation been replaced by the pulmonary conus of the right ventricle, which lies medially to it and the left mid cardiac border is therefore straight. The anterior part of the right ventricle forms the lower portion of the left cardiac border. The apex is rounded.

**Left Oblique View**—Compare with normal Plate 2

Normally the border of the left auricle adjacent to the left bronchus is a straight line and the left auricle is of a triangular peaked shape. In here, the border of the left auricle adjacent to the left bronchus is curved due to enlargement, thus deforming the normal triangular shape of the left auricle. The left bronchus is elevated and compressed by the enlarged left auricle.

**Right Oblique View**—Compare with normal Plate 3

The normally straight esophagus is indented and curved by the enlarged left auricle.

**Summary**—A case of inactive mitral stenosis is presented to demonstrate characteristic roentgenographic findings: straightening of the left mid cardiac contour; elevation and compression of the left bronchus; indentation and curving of the esophagus.

# PLATE 9 INACTIVE MITRAL STENOSIS



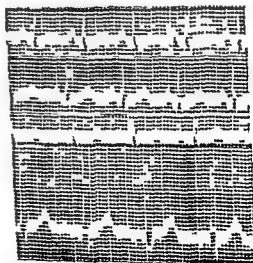
Pulmonary contour is straight (normally concave) due to slight counterclockwise rotation of the heart by the enlarged left auricle



Left oblique view. Left bronchus elevated and compressed by the enlarged left auricle. The border of the left auricle is curved



Right oblique view. Barium-filled esophagus indented and curved by the enlarged left auricle. Note pulmonary conus bulge on the left border



P waves peaked relatively high auricular hypertrophy. Normal electrical axis

## PLATE 10 MITRAL STENOSIS OF MARKED DEGREE

V P, male, white, aged 28

Rheumatic fever followed by heart disease, twelve years previously (1931), at the age of 16. At the time the roentgenogram and electrocardiogram were taken (April 27, 1943), the patient's complaints were palpitation, insomnia, slight gastrointestinal distress. Heart apex displaced to left and down due to cardiac rotation. Diastolic murmur in the mitral area, auricular fibrillation. In September, 1945 the patient developed bacterial endocarditis for which large doses of penicillin were given. Fever, chills, and later petechiae and nodules on extremities. A month later, cerebral embolism, unconsciousness, right hemiplegia, with motor aphasia, facial paralysis, and impaired vision in the right eye. In November, he developed acute cardiac failure and was not expected to live. In January, 1946, he left the hospital markedly improved and at present (December, 1946) is doing fairly well.

**Roentgenogram**—The left auricle is markedly enlarged and appears in the anterior view as an area of increased density in the middle portion of the right cardiac contour. Above it is the ascending aorta, below the right auricle. The left border of the enlarged left auricle would be, if visible, slightly to the left of the sternum, but would not extend all the way to the left cardiac border (see Plate 8). The enlargement of the left auricle while extending to the right and anteriorly causes rotation of the heart counterclockwise on its vertical axis as in the right oblique position (see Plate 3). The aortic knob, pulmonary artery, left ventricular border and apex disappear posteriorly due to rotation. The present left cardiac contour is formed by straight aorta, prominent pulmonary cone of the right ventricle and the hypertrophied right ventricle anterior portion.

**Summary**—A case of marked mitral stenosis is presented to demonstrate on the roentgenogram the enlarged left auricle appearing on the right cardiac contour and the counterclockwise rotation of the heart.

# PLATE 10 MITRAL STENOSIS OF MARKED DEGREE

Ascending aorta

Left auricle

Right auricle

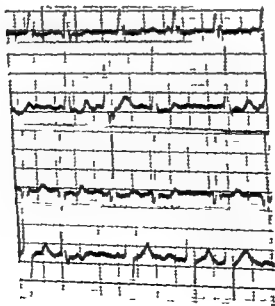
Pulmonary conus of the right ventricle

Right ventricle and right auricle



Heart markedly rotated counterclockwise as in right oblique position  
Enlarged left auricle seen on the right side

Due to rotation aortic knob, left ventricular border apex disappeared posteriorly and replaced by straight aorta, pulmonary conus and anterior portion of the right ventricle



Auricular fibrillation



## PLATE 11 : ELECTROCARDIOGRAM IN MITRAL STENOSIS AND AURICULAR FIBRILLATION

P T, male, white, aged 21. Admitted on July 28, 1941, died on Oct. 26 1941, of advanced heart failure.

Chorea at 11 years of age followed by heart disease. Three years later patient developed dyspnea on exertion, palpitation, precordial pain. Examination on April 25, 1935. Heart regular, enlarged to the left the left heart border in midaxillary line in the sixth interspace. Diastolic thrill over the apex, presystolic rumble heard in the same area. Blood pressure 140/59. Electrocardiogram P notched wide or high suggestive of auricular hypertrophy and abnormal auricular conduction (auriculitis) also right axis deviation indicating right ventricular hypertrophy. The clinical and electrocardiographic findings were about the same as above on March 11, 1937 and May 1, 1939. The patient returned on Oct. 11, 1939 with auricular fibrillation, mitral diastolic murmur, and advanced heart failure. Admitted to the hospital with right hydrothorax, markedly enlarged liver, ascites, edema of lower extremities. Died three months later. No autopsy.

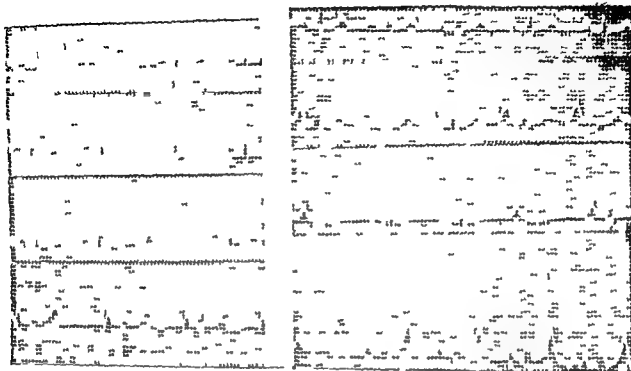
The presence of auriculitis may be revealed in the electrocardiogram by changes in P waves due to impaired auricular myocardial conduction or by abnormal initiation and conduction of impulses, as auricular premature systoles, paroxysmal auricular tachycardia (which is a succession of numerous auricular premature systoles), auricular fibrillation or flutter suggestive of irritable muscle.

**Summary**—A case of mitral stenosis with characteristic electrocardiograms is presented. Etiological diagnosis: Rheumatic fever (chorea) in 1931. Anatomical diagnosis: Mitral stenosis (valvulitis) and auriculitis. Physiological diagnosis: Mild cardiac insufficiency with impaired auricular myocardial conduction in 1934 followed by auricular fibrillation and advanced cardiac insufficiency in 1939 and death two years later.

# PLATE 11 ELECTROCARDIOGRAM IN MITRAL STENOSIS AND AURICULAR FIBRILLATION

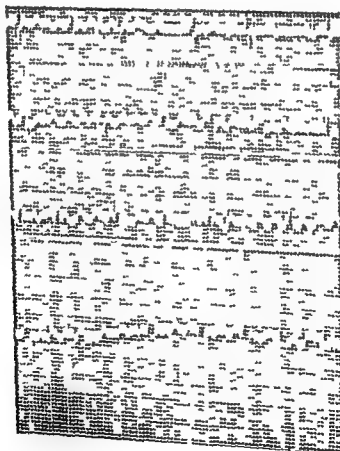
4 a/3a Aged 15

3/11/2 Aged 1



1 notched wide or high-auricular hypertrophy with abnormal auricular conduction  
1 left axis deviation

11 09/23 Aged 13



Auricular fibrillation Right axis deviation

## 2 AORTIC INSUFFICIENCY

The function of a normal aortic valve is (1) to close during the ventricular diastole in order to prevent the blood from flowing back from the aorta into the left ventricle, the valve closes by the pressure of the blood column in the aorta, and the closure causes the normal second aortic sound, which is therefore diastolic in time, (2) to open during the ventricular systole in order to let the blood flow from the left ventricle into the aorta.

The anatomical changes in aortic valvulitis manifest themselves clinically by appearance of a murmur. This murmur will be diastolic in time, as the insufficient valve cannot close well enough during the ventricular diastole in order to prevent the blood from regurgitating from the aorta into the left ventricle. The diastolic murmur is usually soft and frequently heard best in the left third interspace near the sternal border. The aortic soft diastolic murmur is often accompanied by a functional soft diastolic murmur in the mitral area, Austin Flint murmur, not a rumble as in mitral stenosis. At times it may be difficult to decide if the mitral diastolic murmur is not caused by an associated mitral stenosis. In that case, to the characteristic roentgenographic changes of left ventricular hypertrophy due to aortic insufficiency and shown in the anterior and left oblique views, may be added hypertrophy of the left auricle, which indents and curves the esophagus in the right oblique view. The roentgenogram, electrocardiogram, and blood pressure changes may well be correlated with clinical findings to decide whether the aortic insufficiency or mitral stenosis, if both are present, is predominant.

Besides the aortic diastolic murmur, aortic insufficiency has a characteristic pulse sharply rising sharply peaked and abruptly dropping.

Anatomical changes in aortic insufficiency are soon adjusted or compensated by physiological adjustment of the blood pressure and anatomical hypertrophy of the left ventricle. The systolic pressure, i.e., the power of the heart, is slightly elevated in order to propel into

the aorta during ventricular systole the normal volume of blood, which came from the left auricle plus the regurgitated blood during the preceding diastole. The diastolic pressure, i.e., the peripheral resistance is usually markedly depressed, not so much due to regurgitation, as to a marked vasodilatation frequently associated with aortic insufficiency. The blood pressure change is so striking, that when found, it should lead one to suspect aortic insufficiency.

The left ventricular hypertrophy can well be shown on the roentgenogram, anterior and left oblique views (see Plate 12). The hypertrophy is in direct proportion to the insufficiency of the valve, the more the valve is insufficient, the more blood is regurgitated and the more hypertrophy is caused. The average thickness of the left ventricular myocardium is about 12 to 14 millimeters. The myocardium may increase in size to a certain degree, perhaps to about twice the average size, after which the left ventricle begins to dilate, and fail. The roentgenogram in dilatation will show that the left cardiac border is not only rounded but extends far out to the left in the anterior view and markedly overlaps the spine in the left oblique view.

The electrocardiogram in addition to the roentgenogram may furnish evidence of left ventricular hypertrophy, left axis deviation or left axis deviation with high QRS or left ventricular strain, i.e., left axis deviation with high QRS, depressed ST<sub>1</sub>, and deeply inverted T<sub>1</sub> and often T<sub>2</sub>.

As long as the aortic insufficiency is well adjusted or compensated by hypertrophy of the left ventricle and blood pressure changes, the aortic insufficiency, if uncomplicated, is an anatomical residue of an old, healed rheumatic heart disease, well adjusted, and therefore as such does not require any treatment. When a breakdown of compensatory mechanism occurs, dilatation and cardiac failure with impaired function of the left ventricle develop as dyspnea on exertion, orthopnea, and later attacks of paroxysmal nocturnal dyspnea, and if not controlled in time may soon lead to right ventricular failure and death.

## PLATE 12 INACTIVE RHEUMATIC AORTIC INSUFFICIENCY AND MITRAL STENOSIS

A B male white, aged 23

*Streptococcus* sore throat at age 15, followed later by the appearance of heart murmurs. Patient has been well and active since. Mild soft diastolic murmur at the third left interspace near sternal border, mild presystolic rumble at the mitral area not transmitted. Pulsating and dilated blood vessels of the neck and extremities. Corrigan pulse. Blood pressure 140/70.

**Roentgenograms** show evidence of mild hypertrophy of the left ventricle due to aortic insufficiency and of the left auricle due to mitral stenosis. The hypertrophy of the left ventricle is the predominant feature in the roentgenogram and correlates well with predominance of aortic insufficiency with pulse and blood pressure changes clinically. The hypertrophy involves the inflow and outflow tracts of the left ventricle (see Plate 15 for illustration of both tracts). The inflow tract is the posterior portion of the left ventricle with blood flowing in from the left auricle to the apex of the left ventricle. The outflow tract is the anterior portion of the left ventricle with blood flowing out from the apex of the left ventricle into the aorta.

### **Anterior View**—(Compare with normal Plate 1)

The left ventricle outflow tract is elongated, the left cardiac border instead of being straight is rounded and extends straight down below the diaphragm.

### **Left Oblique View**—(Compare with normal Plate 2)

The left ventricle inflow tract is hypertrophied, the left cardiac border near the spine instead of being slightly curved as a bow is rounded and the heart is therefore globular in shape instead of being pear-shaped normally.

The normal electrocardiogram in this case suggests absence of myocardial involvement.

**Summary**—A case of inactive rheumatic aortic insufficiency and mitral stenosis is presented to demonstrate characteristic roentgenographic findings of left ventricular hypertrophy.

# PLATE 12 INACTIVE RHEUMATIC AORTIC INSUFFICIENCY AND MITRAL STENOSIS



Anterior view aortic insufficiency Left ventricle (out flow tract) elongated Left cardiac contour rounded and extends straight down below the diaphragm

Left oblique view aortic insufficiency Hypertrophy of the left ventricle posteriorly (inflow tract) increased rounding of the left cardiac contour the heart is globular in shape



Right oblique view mitral stenosis Prominent pulmonary conus on the left border Enlarged left auricle on the right border



Normal

## PLATE 13 RHEUMATIC CARDITIS WITH AORTIC INSUFFICIENCY AND CARDIAC FAILURE

C B, male, white, aged 16 First admitted on Oct 19, 1945, died on Oct 23, 1945

Acute rheumatic fever at 6½ years of age, for which he was hospitalized for a short time. Recrudescence of rheumatic activity three months later, with three months' hospitalization. At that time the sedimentation rate was 29 mm in one hour, pistol shot femoral pulse, loud diastolic murmur over the right base and fourth left interspace near sternum, diagnosed as aortic regurgitation. In July, 1942 at the age of 12½ years the patient entered the hospital with severe abdominal pain and fever and suspected appendicitis, but later it was interpreted as being due to an acute pericarditis with effusion. Patient was acutely ill for weeks. The course during the next three years was fair, up until the last hospital admission. On admission the apical beat was diffuse and prominent to palpation, the heart markedly enlarged, the left heart border reaching the midaxillary line, there were loud double murmurs in aortic and mitral areas, a Corrigan pulse, blood pressure, 130/20. The liver extended three fingerbreadths down and was tender to palpation. Edema of the legs, ankles, and feet.

Electrocardiograms A and B were practically within normal limits thus ruling out any myocardial involvement. The roentgenogram at that time showed rounding of the left cardiac border due to left ventricular hypertrophy as seen in aortic insufficiency.

Electrocardiogram C, taken during recovery from pericarditis. T<sub>1</sub> is inverted probably due to the pericarditis.

Electrocardiogram D, taken less than a month after (C), showed a normal upright T<sub>1</sub>.

Electrocardiogram E taken four days before death showed a marked left axis deviation, with high, wide slurred QRS. T<sub>1</sub> inverted and in correlation with the x-ray and clinical findings was interpreted as left ventricular strain.

Roentgenogram —The left cardiac border was not only markedly rounded as in hypertrophy, but was far out to the left near the axillary line due to marked hypertrophy and dilatation of the left ventricle. Compare with Plate 12 anterior view, showing left ventricular hypertrophy only.

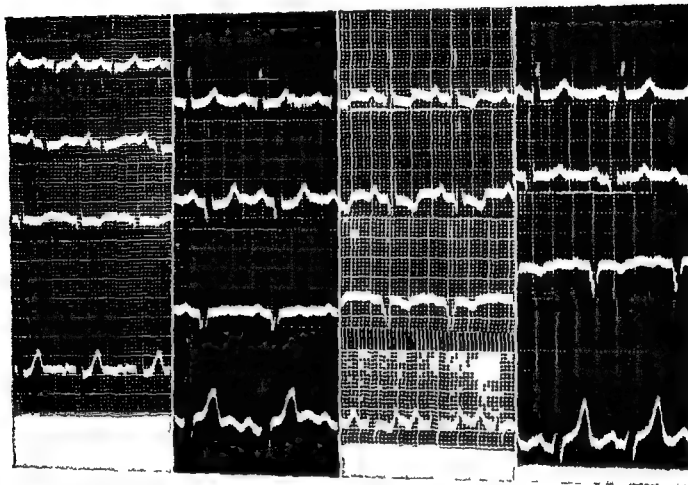
# PLATE 13 RHEUMATIC CARDITIS WITH AORTIC INSUFFICIENCY AND CARDIAC FAILURE

A 3/15/8

B 8/6/30

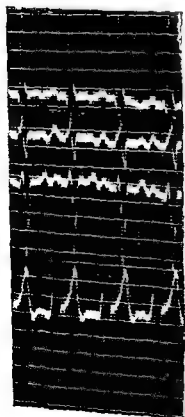
C 11/10/41

D 12/4/41



E 10/19/45 Aged 16

10/19/45



Left ventricle



## PLATE 14 RHEUMATIC CARDITIS WITH AORTIC INSUFFICIENCY, AND CARDIAC FAILURE (CONTINUED)

C. B., male, white, aged 16

**Autopsy**—Chronic aortic endocarditis, marked aortic insufficiency, marked cardiac hypertrophy, old adherent fibrous pericarditis. The heart fills up to one half of the thoracic cavity and weighs 1575 grams.

Chronic passive venous congestion, chronic passive hypertrophy of the liver (nutmeg) ascites, massive edema of the legs and feet. Bronchopneumonia, compression atelectasis of the left lung by the enlarged heart.

**Summary**—A case of rheumatic aortic insufficiency with advanced cardiac failure is presented. The anatomical changes in aortic valvulitis manifest themselves clinically by appearance of aortic diastolic murmur and were soon adjusted or compensated by physiological adjustment of the blood pressure (130/90) and anatomical hypertrophy of the left ventricle. After a period of six years of relative well being, the patient developed acute pericarditis with effusion and was acutely ill for weeks. The patient's course during the next three years was run up until his last hospital admission with advanced left and right ventricular failure, and he died four days later. Autopsy revealed a cor bovinum; the heart weighed 1575 grams while a normal heart would weigh about 250 grams.

The marked aortic insufficiency caused a marked hypertrophy of the left ventricle which reached extreme limits and was followed by a left and later by a right ventricular dilatation and failure. Dyspnea, cyanosis, edema of the legs, ascites, swelling of the liver. In this case the cardiac failure and death were unavoidable results of a complete breakdown of physiologic processes due to a marked aortic insufficiency which could not be well compensated for a long period of time even by the marked left ventricular hypertrophy. Acute pericarditis with effusion was probably a precipitating factor in this physiological breakdown.

**PLATE 14 RHEUMATIC CARDITIS WITH AORTIC INSUFFICIENCY AND CARDIAC FAILURE (CONTINUED)**



Cos bovinum (150 grams) Aortic insufficiency hypertrophy and dilatation of the left ventricle myocarditis adhesive pericarditis

## PLATE 14 RHEUMATIC CARDITIS WITH AORTIC INSUFFICIENCY, AND CARDIAC FAILURE (CONTINUED)

C. B., male, white, aged 16

**Autopsy**—Chronic aortic endocarditis, marked aortic insufficiency, marked cardiac hypertrophy, old adherent fibrous pericarditis. The heart fills up to one half of the thoracic cavity and weighs 1575 grams.

Chronic passive venous congestion, chronic passive hypertension of the liver (nutmeg), scites, massive edema of the legs, ankles and feet. Bronchopneumonia, compression atelectasis of the left lung by the enlarged heart.

**Summary**—A case of rheumatic aortic insufficiency with advanced cardiac failure is presented. The anatomical changes in aortic valvulitis manifest themselves clinically by appearance of aortic diastolic murmur, and were soon adjusted or compensated by physiological adjustment of the blood pressure (130/80) and anatomical hypertrophy of the left ventricle. After a period of six years of relative well being, the patient developed acute pericarditis with effusion and was unable to get up for weeks. The patient's course during the next three years was fair up until his last hospital admission with advanced left and right ventricular failure, and he died four days later. Autopsy revealed a cor bovinum, the heart weighing 1575 grams while a normal heart would weigh about 250 grams.

The marked aortic insufficiency caused a marked hypertrophy of the left ventricle which reached extreme limits and was followed by a left and later by a right ventricular dilatation and failure, dyspnea, cyanosis, edema of the legs, scites, swelling of the liver. In this case the cardiac failure and death were unavoidable results of a complete breakdown of physiological processes due to a marked aortic insufficiency which could not be well compensated for a long period of time even by the marked left ventricular hypertrophy. Acute pericarditis with effusion was probably a precipitating factor in this physiological breakdown.

PLATE 14 RHEUMATIC CARDITIS WITH AORTIC INSUFFICIENCY AND CARDIAC FAILURE (CONTINUED)



Cor bovinum (15.6 grams) Aortic insufficiency hypertrophy and dilatation of the left ventricle myocarditis adhesive pericarditis

## PLATE 15 INACTIVE MITRAL AND AORTIC STENOSIS

D S, male, white, dentist, aged 73

Admitted on June 6, 1944, for prostatectomy, performed four days later  
Died on June 19, 1944, of aspiration pneumonia

The heart weighed with aortic arch, pulmonary artery, and its branches and of the lungs, esophagus and greater part of the thoracic aorta 1050 grams. It is estimated that the heart itself without these other structures probably weighed 600 grams or more.

Longitudinal section of the heart rotated clockwise and placed in left oblique position, the same as in left oblique view on the roentgenogram (see Plate 2). Pathological changes in various chambers of the heart are all shown in this one section: mitral stenosis with dilated left auricle and hypertrophied right ventricle (4 to 10 mm. normally, 3 to 4 mm.), also aortic stenosis with hypertrophied left ventricle (20 mm., normally 12 to 14 mm.). Both inflow and outflow tracts of the left ventricle are shown: the blood flows from the left auricle into the left ventricle through the inflow tract (posterior section of the left ventricular cavity, from mitral valve to apex) and then through the outflow tract (anterior section from apex to aortic valve) into the aorta.

**Summary**—A case of inactive mitral and aortic stenosis is presented. Longitudinal section of the heart demonstrates mitral stenosis with dilated left auricle and hypertrophied right ventricle; aortic stenosis with hypertrophied left ventricle and also inflow and outflow tracts of the left ventricle.



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PLATE 16 MITRAL, TRICUSPID, AND AORTIC STENOSIS  
WITH CARDIAC FAILURE



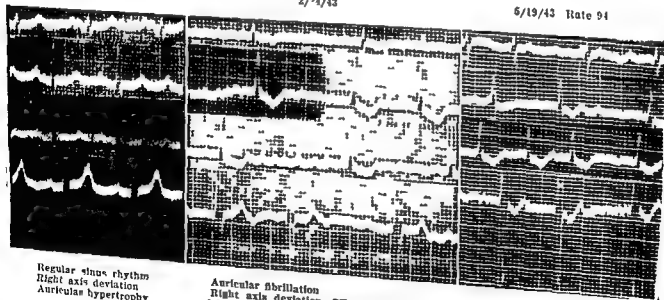
1 19/43 Heart rotated counterclockwise as in mitral stenosis

5/26/43 Rotated heart markedly enlarged as in cardiac failure. Increased shadow density in the right base due to pulmonary infarct and hydrothorax

1/1 /43 Aged 3

2/24/43

5/19/43 Rate 94



Regular sinus rhythm  
Right axis deviation  
Auricular hypertrophy

Auricular fibrillation  
Right axis deviation  
right ventricular strain

ST<sub>2</sub> : depressed (digitalis?) T<sub>2</sub> : Inverted suggesting



### 3 MIXED VALVULITIS

#### PLATE 16 MITRAL, TRICUSPID, AND AORTIC STENOSIS WITH CARDIAC FAILURE

L T, female, white, aged 37 Admitted on March 2, 1943, died on June 1, 1943

Patient had scarlet fever at age of 7, but never rheumatic fever "Heart trouble" at the time of second pregnancy in 1933 She had felt perfectly well until about five months before her present admission her complaints were of gradual onset Complaints on admission were dyspnea, palpitation, chest pain, productive cough, ankle edema, anorexia, and indigestion Heart enlarged, the left border in the anterior axillary line, auricular fibrillation, loud systolic and diastolic murmurs, most marked at the apex, heard over entire precordium Blood pressure 170/80 The liver was four finger breadths below the right costal margin Spleen not palpable

Her course in the hospital was one of progressive cardiac failure, marked by periods of disorientation and confusion, which began on the second hospital day She developed ascites and hydrothorax and these fluids were drained several times She had frequent episodes of fever and coughing, with frothy, blood tinged sputum from time to time—pulmonary infarcts

**Roentgenograms**—Heart markedly enlarged and rotated counterclockwise The normal left cardiac border consisting of aortic knob pulmonary artery and left ventricle disappeared posteriorly The upper portion of the left cardiac border is a straight vertical line The middle portion which is usually concave is a straight line due to prominence and hypertrophy of the pulmonary conus of the right ventricle The lower portion is the anterior part of the right ventricle

A roentgenogram taken on May 26, 1943, shows the enlarged left auricle on the right cardiac border and the heart markedly enlarged as in cardiac failure

**Electrocardiogram**—Regular sinus rhythm slight axis deviation auricular hypertrophy, replaced later by auricular fibrillation and right ventricular strain

**PLATE 17 MITRAL, TRICUSPID, AND AORTIC STENOSIS  
WITH CARDIAC FAILURE (CONTINUED)**



Mitral stenosis Hypertrophy and dilatation of the left auricle



Tricuspid stenosis Dilated and hypertrophied right auricle Dilated and hypertrophied right ventricle due to mitral stenosis



Aortic stenosis Left ventricular hypertrophy the wall is 17 mm in thickness Note a calcified nodule about 2 cm below the aortic valve

## PLATE 17 MITRAL, TRICUSPID, AND AORTIC STENOSIS WITH CARDIAC FAILURE (CONTINUED)

L 1 female, white, aged 37

**Autopsy**—The chief change in the heart is valvular most marked at the mitral valve. Chronic inducive mitral aortic, and tricuspid endocarditis with extensive calcification and stenosis of aortic and mitral valves. Pulmonary valve is unaltered. Hypertrophy and dilatation of left and right ventricles and auricles. Chronic passive hyperemia of the lungs (brown induration) liver (nutmeg), spleen and other abdominal viscera bilateral hydrothorax (right 1500 cc left 100 cc), ascites (5000 cc). Thrombosis of pelvic veins, mural thrombi of both auricular appendages multiple pulmonary emboli with multiple acute and organizing infarcts of the lungs and infarcts (early or in progress) of the spleen and kidneys. The multiple pulmonary infarcts could have arisen from the right auricular thrombus and those in the spleen and kidneys from the left auricular thrombus.

Sparsely disseminated subacute peritonitis of the myocardium. Disseminated subacute focal inflammation of the liver (portal spaces).

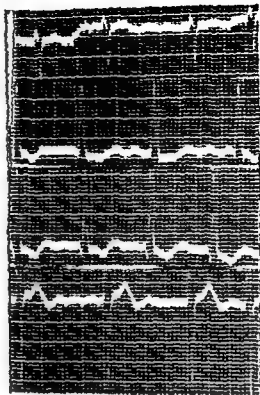
The interpretation of the post mortem findings are consistent with the primary disease of chronic rheumatic valvular heart disease with evidence of left and right ventricular failures as evidenced by the chronic passive hyperemia of the lungs on the one hand and of the liver and other viscera the hydrothorax and ascites on the other. The only evidence of disseminated inflammation in the body suggestive of a rheumatic peritonitis is found in the myocardium and liver but because of the absence of significant inflammation elsewhere the significance of the peritonitis of the myocardium without characteristic Aschoff nodules is not interpreted as conclusive evidence of recurrent rheumatic inflammation.

**Summary**—A case of rheumatic heart disease with involvement of mitral tricuspid and aortic valves with predominance of mitral stenosis and advanced heart failure is presented.

PLATE 18 VALVULITIS OF ALL THE VALVES, CARDIAC FAILURE



3 20 43 Unusually prominent pulmonary conus suggests pulmonary stenosis



2/0/43 Aged 20 Male 6 Atrial fibrillation Right ventricular strain Right axis deviation Tz a inverted



Mitral stenosis Dilated and hypertrophied left auricle



Aortic insufficiency Dilated and hypertrophied left ventricle

## PLATE 18 VALVULITIS OF ALL THE VALVES, CARDIAC FAILURE

W S, male, white, aged 20 Admitted on March 19 1943, died on April 20, 1943

Rheumatic fever at the age of 4 hospitalized for one year He was well until the age of 10 when he had another severe attack of polyarthritis and precordial pains A third attack of arthritis occurred at the age of 15 and lasted six months He was well until the present hospital admission Although he had tonsillectomy at the age of 4 he had frequent sore throat his night ear had drained for the previous seven years

On admission patient complained of dyspnea of two months duration, edema of legs for one month Physical findings revealed orthopnea, cyanosis, normal temperature, bilateral perforated eardrums without discharge, carious teeth Heart enlarged to right and left, systolic and diastolic thrills and murmurs over entire precordium Auricular fibrillation as in mitral stenosis Blood pressure 175/45 as in aortic insufficiency Edema of the legs, sacral edema, tense abdomen, enlarged pulsating liver, as in tricuspid stenosis

The course in the hospital was one of progressive heart failure marked early by development of pleuritic pain, anorexia and vomiting Temperature for the previous two weeks between 100 and 102° F In the last week he developed thrombosis of the left femoral vein

**Roentgenogram**—The heart is enlarged and rotated counterclockwise The enlarged left auricle is seen on the right cardiac border as increased shadow density Due to counterclockwise rotation of the heart the aortic knob disappeared posteriorly and the lower left cardiac border is formed by the anterior position of the hypertrophied right ventricle The unusually prominent pulmonary conus on the mid portion of the left cardiac border suggests marked hypertrophy of the conus as in pulmonary stenosis

**Electrocardiogram**—Auricular fibrillation Right ventricular strain as in advanced mitral and pulmonary stenosis

PLATE 19 VALVULITIS OF ALL THE VALVES, CARDIAC  
FAILURE (CONTINUED)



Pulmonary stenosis Right ventricle including pulmonary cone is markedly hypertrophied



Pulmonary stenosis Hypertrophy of the right ventricle



Tricuspid stenosis Dilated and hypertrophied right auricle



Fibrous adhesive pericarditis

## PLATE 19 VALVULITIS OF ALL THE VALVES, CARDIAC FAILURE (CONTINUED)

W S, male, white, aged 20

**Autopsy**—Chronic, indurative mitral, aortic, tricuspid and pulmonary endocarditis with stenosis of mitral, pulmonic and tricuspid valves and insufficiency of the aortic valve. Hypertrophy and dilatation of all cardiac chambers, especially of the left and right ventricles and the left auricle. Fibrous adhesive pericarditis.

Chronic passive hyperemia of the lungs, liver, and abdominal viscera. Recent thrombosis of the veins of the left lower extremity, extending to the bifurcation of the inferior vena cava. Sparse pulmonary embolism—thrombi in each branch of pulmonary artery with small disseminated hemorrhagic pulmonary infarcts. Moderate anasarca of the left lower extremity, scrotum and sacrum, slight anasarca of the right foot. Little changes of the liver, cloudy swelling of the kidneys. Slight icterus. Bilateral disseminated fibrous pleuritis. Sparse, acute, hemorrhagic bronchopneumonia.

**Summary**—A case of rheumatic heart disease with involvement of all the valves, including pulmonic, fibrous adhesive pericarditis and advanced heart failure is presented.

The roentgenogram is characteristic of advanced mitral stenosis, but the unusually prominent pulmonary conus on the left cardiac border is suggestive also of pulmonary stenosis in addition to mitral stenosis.

The electrocardiogram with auricular fibrillation, as in mitral stenosis and right ventricular strain correlate well with roentgenographic and clinical findings of advanced right heart failure.

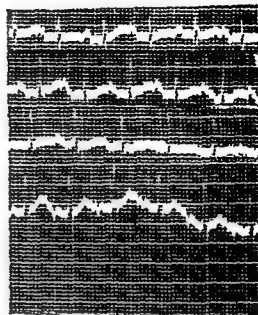
The autopsy showed the valves most involved were mitral and pulmonic (stenosis), with aortic insufficiency (blood pressure 175/40) and slight tricuspid stenosis next, and also characteristic changes in all the chambers of the heart.

PLATE 20 ACTIVE CARDITIS, WITH PREDOMINANT  
MYOCARDITIS, AND CARDIAC FAILURE

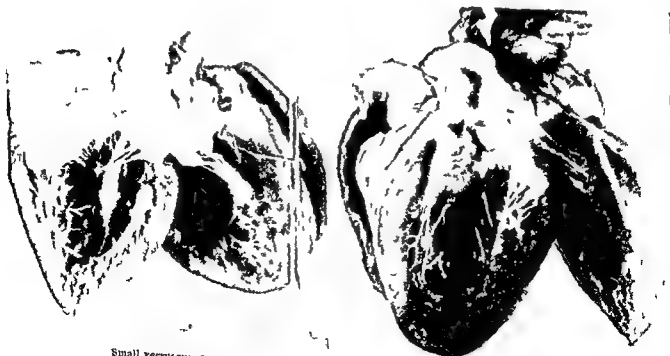
9/11/41 Aged 6



Heart enlarged in all directions



Rate 120 PR 0.20 second  
Prolonged PR interval Incomplete VV  
block  
All T's are low and with sinus tachycar  
dia suggest myocardial insufficiency



Small verrucous vegetations on the mitral valve No significant aortic changes Dilatation of the left ventricle due to myocarditis Minimal fibrinous pericarditis



## 4 MYOCARDITIS

### PLATE 20 ACTIVE CARDITIS, WITH PREDOMINANT MYOCARDITIS, AND CARDIAC FAILURE

P V, female, white, aged 6 Admitted on July 30, 1941, died on Oct 22, 1941

Swollen and painful wrists and ankles, five months before admission Pain and swelling in the joints of the hands and abdominal pain with occasional vomiting of one month's duration Physical examination on admission revealed a poorly nourished child Pulse 116 respirations 20 blood pressure, 105/70 Decayed teeth, enlarged and reddened tonsils cervical adenopathy Heart enlarged to right and left palpable systolic thrill loud blowing systolic apical murmur, transmitted to axilla (mitral insufficiency) Liver enlarged one fingerbreadth below the costal margin Swollen fingers and ankles Sedimentation rate 34 millimeters

The course in the hospital was one of gradually progressive heart failure with enlargement of the heart and liver with edema A month after admission the heart was enlarged to the anterior axillary line and a loud squeaking pericardial friction rub was heard at the apex

**Röntgenograms** taken on various dates showed moderate diffuse cardiac enlargement suggesting pericarditis or dilatation due to myocarditis

With a weak flabby myocardium the **electrocardiogram** showed prolonged PR interval and low voltage of all T's

**Autopsy**—Subacute rheumatic mitral and tricuspid endocarditis Subacute rheumatic myocarditis with pale flabby pinkish gray myocardium of both ventricles and dilatation of the cardiac chambers (but without Aschoff bodies microscopically) Minimal fibrinous pericarditis Petechial hemorrhages of the epicardium pleura and lining of the stomach Hydrothorax ascites meningitis Chronic passive hyperemia of the lungs liver spleen and kidneys Marked fatty changes of the liver

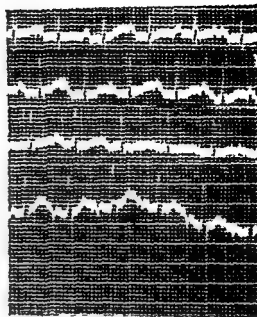
**Summary**—A case of active carditis with predominant myocarditis and cardiac failure is presented

# PLATE 20 ACTIVE CARDITIS, WITH PREDOMINANT MYOCARDITIS, AND CARDIAC FAILURE

9/11/41 Aged 6



Heart enlarged in all directions



Rate 130 PR 0.20 second  
Prolonged PR interval incomplete AV  
block  
All T's are low and with sinus tachycar  
dia suggest myocardial insufficiency



Small verrucous vegetations on the mitral valve No significant aortic changes Dilatation of the left ventricle due to myocarditis Minimal fibrinous pericarditis

## PLATE 21 ELECTROCARDIOGRAM IN MYOCARDITIS (COMPOSITE ILLUSTRATION)

Myocardial damage is an anatomical and not a cardiographic diagnosis. The electrocardiogram shows only changes in conduction, which, considering the clinical findings, may lead us to suspect myocardial damage.

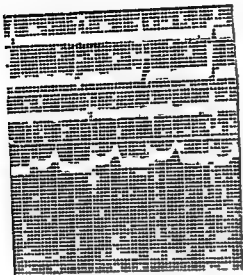
The walls of the auricles (changes in P), ventricles (changes in QRS, ST, T) or the septum (auriculoventricular block, incomplete or complete, or bundle branch block), may be involved. One example of auricular myocardial involvement is that present in mitral stenosis with auriculitis and changes in P which later may lead to auricular fibrillation (see Plate 11). In contradistinction to myocardial infarction, where the myocardial damage is usually localized the rheumatic myocarditis is usually generalized of a disseminated type causing changes in all the leads with disturbance in intraventricular conduction as shown by wide slurred or splintered QRS, deformed ST, or low, inverted T's. An associated myocardial insufficiency may cause low voltage of all leads and tachycardia. If the septum is involved, incomplete block with prolonged PR and dropped beats, complete block or bundle branch block may be present.

Serial electrocardiograms may reveal changes indicating activity of myocardial damage. Usually, rheumatic myocarditis heals without any impairment of myocardial conduction and a normal electrocardiogram is found as indication of complete healing. In some cases the scar tissue may permanently interfere with normal conduction. Inverted T's or various forms of heart block remain permanent and unchanged for years as residue of an old healed damage.

**Electrocardiogram C—D** S. female white with a negative history on May 30, 1924, aged 16 contracted upper respiratory infection with severe sore throat. Temperature 102 F and pulse 100 followed in a few days by attacks of Adams Stokes syndrome with unconsciousness, convulsions and heart rate as low as 15. Acutely ill for a few weeks she recovered and has been well since. The complete auriculoventricular block found in June 1924 remained unchanged for over twenty-two years and was still present in March 1947.

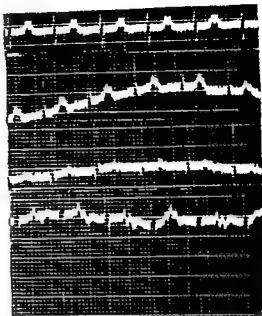
# PLATE 21 ELECTROCARDIOGRAM IN MYOCARDITIS (COMPOSITE ILLUSTRATION)

A 5/15/40 Aged 40



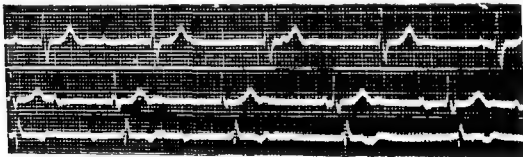
T<sub>1</sub> : low voltage  
T<sub>2</sub> : inverted  
QRS inverted

B 9/7/41 Aged 6



Late P waves 0.4 second Low grade of incomplete block

C 3/6/31 Aged 31



Rate auricular 65 ventricular 46 No relation between auricular (P) and ventricular (QRS T) contractions Complete heart block Present since June 1934 to date (March 1941)

D 10/19/41 Aged 16



In the first electrocardiogram (A) rheumatic myocarditis affects the myocardium in general without affecting the bundle of His

The last three electrocardiograms (B C D) are examples of rheumatic myocarditis affecting the His bundle (special tissue in myocardium) causing either incomplete or complete auriculoventricular block or bundle branch block

## 5 PERICARDITIS

### PLATE 22 PERICARDITIS WITH EFFUSION

A B, male, white, physician, aged 36

Upper respiratory infection and tonsillitis, followed about three weeks later by fever up to  $101^{\circ} F$ , spasmodic cough without expectoration, and dull lower substernal pain

Heart Apex beat neither visible nor palpable heart enlarged to left and right, heart sounds are audible, no murmurs. A loud pericardial rub heard in lower sternal region Blood pressure, 118/78 pulse 140 regular Temperature,  $99.6^{\circ} F$  Patient hospitalized for three weeks Pericardial rub disappeared at end of the first week Patient made an uneventful recovery

**Röntgenograms**—Anterior, left oblique and right oblique views (see Plate 23), demonstrate the absorption of fluid

**Electrocardiograms**—Electrocardiograms taken in this case showed a low voltage of QRS, T, and tachycardia (120 to 140), but normal a month later and ever since

The electrocardiographic abnormalities as low voltage or inversion of  $T_1$  are the valuable clues in doubtful or unsuspected cases of pericarditis. The electrocardiographic changes are probably due to an associated myocarditis but may be due to compression of coronary vessels by pericardial fluid or adhesions and impairment of blood supply to the myocardium. In the majority of cases when recovery takes place  $T$ 's will be found normal again. In others inversion of  $T_1$  may remain permanently as a sign of scar tissue in the myocardium or pericardium which interferes with a normal myocardial conduction or normal blood supply to the myocardium and as such is of no clinical value. Inversion of  $T_1$  in an adult may suggest a possibility of coronary disease. The correlation with clinical findings and history will clarify this point.

**Pathology**—Plates 14, 19 and 20 illustrate the gross pathology of the fibrous adhesive pericarditis as a residuum of rheumatic carditis.

# PLATE 22 PERICARDITIS WITH EFFUSION



4/9/44 Cardiac shadow enlarged in all directions  
Loud pericardial rub heard in the lower sternal region



4/6/44 Cardiac shadow decreased in size Left base  
pleuritis Slight pericardial rub still present



5/5/44 Cardiac shadow near normal Pericardial rub  
gone L. ft base pleuritis present



5/24/44 Cardiac shadow normal Left base pleuritis  
disappeared

## PLATE 23 PERICARDITIS WITH EFFUSION (CONTINUED)

A B, male, white physician aged 36

Roentgenograms taken in left and right oblique views illustrate the extent of pericardial fluid to the left and right posteriorly

**Left Oblique View**—The cardiac shadow is diffusely enlarged and markedly overlaps the spine as in marked left ventricular hypertrophy and dilatation (see Plate 21) Correlation with clinical findings will clarify the differential diagnosis

**Right Oblique View**—The cardiac shadow is diffusely enlarged and obliterates the entire retrocardiac space especially in the lower portion In mitral stenosis (see Plates 9 and 12) the retrocardiac space is only partially compressed in the upper portion by the enlarged left auricle

**Summary**—A case of pericarditis with effusion is presented to demonstrate characteristic roentgenographic findings

PLATE 23 PERICARDITIS WITH EFFUSION (CONTINUED)



4/ 44 Left oblique view Cardiac shadow enlarged posteriorly to left overlapping the spine



44 Left oblique view Cardiac shadow normal



4 44 Right oblique view Cardiac shadow enlarged posteriorly to right obliterating retrocardiac space



5/ 44 Right oblique view Cardiac shadow normal Retrocardiac space is clear



## 5 ACTIVE CARDITIS

### PLATE 24 ACTIVE CARDITIS WITH CARDIC FAILURE AND PNEUMONIA

J R, male, white, aged 16 Admitted on Jan 25, 1944, died on Jan 29, 1944

Apparently well until five months before admission, when he had rheumatic fever with involvement of the heart, from which he apparently recovered He was well until the previous week, when he contracted pneumonia On admission, lungs full of rales and wheezes with decreased breath sounds in the right base, with heart sounds difficult to make out because of the noisy respirations and blowing murmurs obscuring the heart tones Blood pressure 130/70 Liver palpable almost to the navel abdomen distended Sputum disclosed type XIX pneumococcus Patient became progressively worse and died four days after admission

#### Autopsy —

##### 1 Acute and chronic rheumatic carditis

a Acute verrucous and chronic indurative aortic and mitral endocarditis with aortic stenosis and insufficiency mitral insufficiency and minimal indurative tricuspid endocarditis The acute changes are those of minute pinpoint to pinhead sized vegetations The chronic changes include adherence of the two leaflets at the commissures thickening and rolling back and shortening of the margins and leaflets with part of the leaflets held downward by thick tendinous cords

b Acute subacute, and chronic rheumatic myocarditis, with Aschoff nodules

c Acute and organizing serosanguinous and fibrinous pericarditis

d Hypertrophy and dilatation of the cardiac chambers

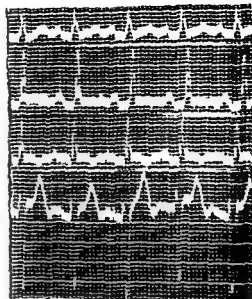
2 Mild bilateral hydrothorax ascites, subacute passive congestion of the abdominal organs mild acute hyperplasia of the spleen

3 Acute pneumococcal lobular pneumonia involving all lobes and mild right fibrinous pleuritis

**Summary**—A case of active carditis with cardiac failure complicated by pneumonia is presented

# PLATE 24 ACTIVE CARDITIS WITH CARDIAC FAILURE AND PNEUMONIA

1/26/44 Aged 16 Rate 110



All QRS slurred QRS inverted T low voltage T inverted

Heart shadow enlarged in all directions Bilateral pneumonitis



Acute verrucous and chronic indurative mitral endocarditis

## 7 BACTERIAL ENDOCARDITIS

### PLATE 25 BACTERIAL ENDOCARDITIS

It is customary on the basis of duration of the infection to distinguish two forms (1) acute as parts of sepsis, resulting from some local infection signs of sepsis usually outweigh those of cardiac involvements (2) subacute superimposed upon an old valvular deformity usually rheumatic or congenital more rarely atherosclerotic or syphilitic

With the advance of penicillin the time element distinguishing the two forms has been markedly changed. Also a number of cases of so called subacute bacterial endocarditis have been of very short duration due to fatal complications as for instance cerebral hemorrhage embolus or ruptured mycotic aneurysm. Therefore it would be logical if clinicians would discard the words acute or subacute in diagnosing bacterial endocarditis.

In addition to septic fever enlarged spleen positive blood culture and cardiac murmur the bacterial endocarditis manifests itself by various vascular phenomena: (1) Emboli, as cerebral or Osler's nodes on fingers and toes (2) Arteritis with thrombosis and mycotic aneurysm as cerebral or coronary blood vessels (3) Hemorrhages as cerebral renal or petechiae. If the above manifestations affect the vital organs they may cause death in spite of successful treatment of the bacterial endocarditis and sterilization of the blood stream with penicillin. The same is true when bacterial endocarditis is complicated by cardiac failure.

## PLATE 25 BACTERIAL ENDOCARDITIS

### SUBACUTE BACTERIAL ENDOCARDITIS

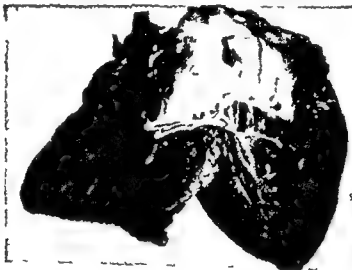
H S male white aged 5° Rheumatic fever at age 1°

Chronic indurative mitral and aortic endocarditis. Acute thrombovegetative post mortem mitral and aortic endocarditis

The mitral leaflets show chronic changes and in addition acute changes consisting of a few small brown and pale rough vegetations. Note small dark patches of subacute bacterial endocarditis disseminated above the margin of the mitral leaflets

The myocardium of the left ventricle is 8 to 10 mm thick. Ectopic pericardial hemorrhages. Thrombosis of the right auricular appendage

Death due to pulmonary infarct and aspiration bronchopneumonia



### ACUTE BACTERIAL ENDOCARDITIS

C P male white aged 66

Cystitis with septicemia. Large infected purulent vegetation on the middle aortic cusp. Abscess of the left lung. Arachnoiditis with meningitis. Septic spleen

## PLATE 26 BACTERIAL ENDOCARDITIS WITH CEREBRAL HEMORRHAGE

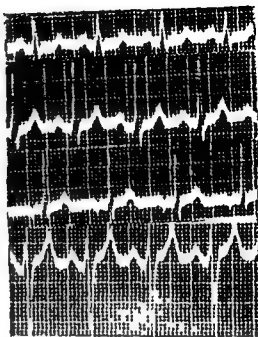
G B female white aged 32 Admitted on July 6, 1943 died on July 29 1943

Old rheumatic aortic insufficiency and mitral stenosis with loud double aortic and mitral murmurs water hammer pulse, blood pressure 140/10 Following tooth extraction, the patient developed bacterial endocarditis with petechiae and positive blood cultures for *Streptococcus viridans* Short duration of sickness due to fatal cerebral hemorrhage

**Autopsy**—Chronic rheumatic mitral and aortic endocarditis with stenosis and insufficiency Streptococcal viridans mitral endocarditis Extensive subarachnoid basal hemorrhage Numerous disseminated hemorrhages of the serosa of the stomach and intestines submucosal hemorrhages of the kidneys hemorrhages of the lungs Acute hyperplasia of the spleen

Note that rheumatic valvulitis affected mitral and aortic valves while bacterial endocarditis affected the mitral valve only

**Summary**—A case of bacterial endocarditis with positive blood cultures for *Streptococcus viridans*, superimposed upon an old rheumatic valvulitis and of short duration due to fatal cerebral hemorrhage is presented



Normal

Left midcardiac contour is straight—mitral stenosis. Left lower cardiac border is out and rounded



Mitral valve



Aortic valve

Chronic rheumatic mitral and aortic endocarditis with stenosis and insufficiency  
Acute *Streptococcus viridans* mitral endocarditis Subarachnoid basal hemorrhage

## PLATE 27 BACTERIAL ENDOCARDITIS WITH MYCOTIC ANEURYSM

A P, male aged 38 Admitted on July 9 1940, died on August 16, 1941, after thirteen months of hospitalization

Old rheumatic mitral and aortic endocarditis Blood pressure, 116/60 Bacterial endocarditis with enlarged spleen, numerous petechial nodules (emboli) on toes and fingers but always negative blood cultures during the thirteen months in the hospital Two months before death, patient developed acute serofibrinous pericarditis

**Autopsy**—Indurative mitral and aortic endocarditis Subacute bacterial mitral endocarditis Mycotic aneurysm and thrombosis of the circumflex branch of the left coronary artery Organizing, obliterative pericarditis The maximum thickness of the wall of the left ventricle at the base is 14 millimeters whereas the apex thins down to 4 millimeters Extensive disseminated subpleural hemorrhages disseminated petechial hemorrhages of the ileum marked hyperplasia of the spleen old healed infarctions of the right kidney

Mycotic embolic aneurysm of a coronary artery results from softening of the arterial wall at the site of lodgment of an embolus secondary to bacterial endocarditis The proximal part of the left coronary is most frequently involved and rupture occurs in about one half of the cases It is usually single one to many centimeters in size (3 cm here) and the lumen may show partial or complete occlusion by a thrombus in various stages of organization

This case reminds us of one autopsied two years ago Male white aged 38 Subacute bacterial endocarditis superimposed upon a chronic indurative mitral endocarditis ruptured mycotic aneurysm of the left posterior cerebral artery with extensive cerebral hemorrhage

**Summary**—A case of bacterial endocarditis with mycotic aneurysm superimposed upon an old rheumatic valvulitis is presented Negative blood cultures during thirteen months of hospitalization

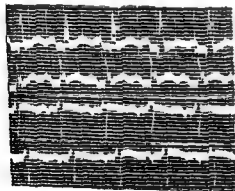
# PLATE 27 BACTERIAL ENDOCARDITIS WITH MYCOTIC ANEURYSM



4/10/41 Left ventricular border is out to the left

7/11/41 Heart markedly enlarged in all directions  
Pericarditis

6 0/41 Aged 36



Left axis deviation QRS notched QRS,  
inverted T; isoelectric possibly a technical  
error as Lead 4 is similar to Lead 3



Indurative mitral and aortic endocarditis Subacute bacterial mitral  
endocarditis Organizing pericarditis Mycotic aneurysm with throm  
bosis of the left coronary artery



## PLATE 28 BACTERIAL ENDOCARDITIS WITH SPONTANEOUS PERFORATION OF RIGHT AORTIC CUSP

T C male, Negro, aged 52 Admitted on Dec 26, 1917, died on March 19 1918

Admitted with complete urinary retention, secondary to benign prostatic hypertrophy unrelieved for two days because of unsuccessful attempts at self catheterization Temperature 100.2 F, blood pressure 190/100 Heart normal no murmurs On the eighth day suprapubic cystotomy was performed The postoperative course can be divided into four periods

1 The first period of sixteen days from the day of cystotomy to the day when *Bacillus coli* appeared on blood culture Septic temperature

2 The second period of twenty two days from the day of positive blood culture to the day of appearance of a loud systolic apical murmur

3 The third period of twenty two days from the day of appearance of heart murmur to the day of cerebral embolism (spastic extremities) Petechiae of the conjunctivae and skin were noted

4 The fourth period of eight days from the day of cerebral embolism to the day of death

**Autopsy**—Slight hyperplasia of the prostate gland fibrous periprostitis unhealed cystotomy wound acute cystometritis

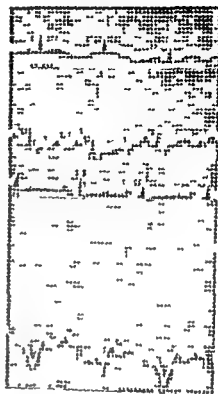
Acute thromboembolitic aortic and mitral endocarditis spontaneous perforation of right aortic cusp Septic infarcts of the spleen left kidney cerebrum and myocardium Petechial hemorrhages of the skin and small intestines Slight bilateral hydrothorax ascites and anasarca Slight generalized arteriosclerosis Hypertrophy of the left ventricle

**Summary**—A case of bacterial (*Bacillus coli*) endocarditis following acute contumacious infection is presented Note septic temperature positive blood cultures appearance of heart murmurs cerebral embolism and infarct petechial hemorrhages of the skin and spontaneous perforation of right aortic cusp

PLATE 28 BACTERIAL ENDOCARDITIS WITH SPONTANEOUS  
PERFORATION OF RIGHT AORTIC CUSP



Normal



T<sub>1</sub> is a low T<sub>1</sub> inverted (normal with old technique of taking Lead 4)



Bacterial mitral and aortic endocarditis following acute genitourinary infection. Vegetations and thrombi on aortic cusps. Perforation of right aortic cusp. Slight chronic adherence of posterior and left cusps. Slight perforation of anterior mitral leaflet. A vegetation on its auricular surface.

### III

## ARTERIOSCLEROTIC HEART DISEASE

### PLATE 29 ATHEROSCLEROSIS OF THE AORTA

The atherosclerotic process involves the aorta, mitral and aortic valves, coronary arteries, and may be associated with abnormal rhythm in the auricles, auricular flutter, or fibrillation.

In the atherosclerosis of the aorta the intima is altered by atheromatous plaques and calcification with increased severity from the arch down toward the abdominal aorta. The weakened wall may dilate, causing a diffuse dilatation and tortuosity of the aorta more frequently so when associated with hypertension. Clinically this condition is unnoticed by the patient and does not cause any symptoms unless rupture of the weakened aortic wall occurs. It may rupture completely and cause a fatal hemorrhage or may rupture partially with dissection of the coats of the vessel by blood. In dissecting aneurysm death may not occur immediately but be postponed for days, weeks or even months. To complete the discussion of aneurysms we included a short description of arteriovenous and aneurysms (Plate 33) which are not due to atherosclerosis.

The atherosclerosis of the mitral valve may cause mitral insufficiency with a soft systolic apical murmur usually of little consequence to the patient.

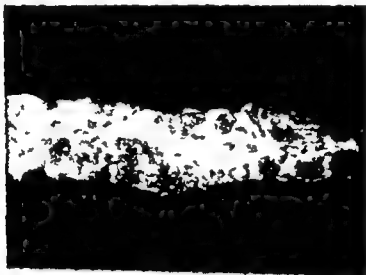
The atherosclerosis of the aortic valve may cause aortic insufficiency with a soft diastolic aortic murmur and characteristic blood pressure changes. The condition and result are in all respects similar to aortic insufficiency of rheumatic etiology and for further details we refer the reader to that section. Fibrosis and sclerosis may cause slight aortic stenosis with soft systolic aortic murmur or nodular calcification, aortic stenosis with pronounced blowing, systolic aortic murmur, small plateaulike pulse, normal or slightly lower blood pressure and marked hypertrophy of the left ventricle.

Atherosclerosis of the coronary arteries will be discussed in detail later.

## PLATE 23 ATHEROSCLEROSIS OF THE AORTA

L D female white aged 5

The lining of the aorta is extensively altered by gray and yellow atheromatous plaques with considerable calcification and with increased severity from arch downward until at the bifurcation there is a partial occlusion by yellow and predominantly red tan mural thrombus. In addition there are numerous small thin reddish brown mural thrombi forming conspicuous soft nodules upon the plaques in thoracic and abdominal aorta.



W A male white aged 65

Ruptured aneurysm of abdominal aorta. Extensive generalized arteriosclerosis including the aorta pulmonary and coronary arteries. Benign nephrosclerosis.

## PLATE 30 ATHEROSCLEROSIS OF THE AORTA, RUPTURED ABDOMINAL AORTIC ANEURYSM

B C, male, white, aged 85 Admitted on Oct 6, 1941, died on Dec 7, 1941

Patient had an aneurysm of abdominal aorta for the preceding three years, according to his physician's statement Four hours before admission patient had a sharp pain in the left lower quadrant slight nausea, desire to defecate, pallor and sweating Two hours later, he felt slightly better Blood pressure, 118/102 pulse of fair quality auricular fibrillation On admission patient appeared acutely ill with pallor of the face and cold extremities Pulse 104 temperature 99.6° F respirations 36 blood pressure, 82/48 A pulsating mass was palpated in the abdomen extending downward to the bifurcation of the aorta, with increased tenderness in the left lower abdomen Three hours after admission patient appeared to be in extreme shock the entire abdomen felt very full as though with fluid and patient died soon after

**Autopsy**—Atherosclerosis of the aorta aneurysm of the lower part of abdominal aorta with mural thrombosis Spontaneous perforation of the aneurysm with retroperitoneal hematoma and hemoperitoneum Atherosclerosis of the coronary arteries slight fibrosis of myocardium minute scars and organizing infarctions of the myocardium The thoracic portion of the aorta is tortuous Beginning at the superior end of the thoracic aorta and extending down along the posterior wall to the level of the diaphragm, at least 90 per cent of the lumen is altered by soft yellowish brown elevations some of which are embedded in ulcerative regions

**Summary**—A case of atherosclerosis of the aorta with ruptured aneurysm of abdominal aorta in a man 85 years of age is presented

# PLATE 30 ATHEROSCLEROSIS OF THE AORTA, RUPTURED ABDOMINAL AORTIC ANEURYSM

Dilated descending aorta seen as a diffuse shadow with a straight border extending from the aortic knob down and to the left of the pulmonary artery. Within this shadow the triangular hilum may be seen.

The interrupted short lines indicate the outline of the pulmonary artery.



Slight dilatation of the heart. Slight adherence of posterior and left aortic cusps. Slight dilatation of ascending aorta. Atherosclerosis of thoracic and abdominal aorta. Aneurysm with mural thrombus of lower part of abdominal aorta. Spontaneous perforation of the aneurysm. Hemoperitoneum.



## PLATE 31 SCLEROSIS OF THE ABDOMINAL AORTA

Roentgenograms of the spine and pelvis anterior and lateral views visualize the sclerosis of the abdominal aorta and iliac arteries.

Chest roentgenograms anterior and left oblique views, visualize the aortic arch and the thoracic aorta.

In anterior view (Plate 1) the ascending aorta is seen on the right upper border while the aortic knob is on the left. If aorta is dilated and tortuous, the ascending aorta on the right is bulging the aortic knob may be prominent with calcification noted on the border the descending aorta is a diffuse shadow with a straight border extending from the aortic knob down and to the left of the pulmonary artery. Frequently the tortuous course of the thoracic aorta may be followed within the cardiac shadow down to the diaphragm.

In left oblique view (Plate 2) the aortic arch is seen over the left triangle and bifurcated trachea. The dilatation of the aortic arch is plainly noticeable on the left oblique view as a wide round shadow over the heart and overlapping the spine (see Plate 51).

# PLATE 31 SCLEROSIS OF THE ABDOMINAL AORTA



Anterior view Sclerosis of pelvic arteries and those of the thighs

Lateral view Marked sclerosis of the entire abdominal aorta

G S male white aged 61

Marked osteoarthritic lipping in the bodies of the lumbar vertebrae Decalcification in the bones of the pelvis and lumbar vertebrae



## PLATE 31 SCLEROSIS OF THE ABDOMINAL AORTA

Roentgenograms of the spine and pelvis, anterior and lateral views, visualize the sclerosis of the abdominal aorta and iliac arteries.

Chest roentgenograms, anterior and left oblique views, visualize the aortic arch and the thoracic aorta.

In anterior view (Plate 1) the ascending aorta is seen on the right upper border, while the aortic knob is on the left. If aorta is dilated and tortuous, the ascending aorta on the right is bulging, the aortic knob may be prominent with calcification noted on the border. The descending aorta is a diffuse shadow with a straight border extending from the aortic knob down and to the left of the pulmonary artery. Frequently the tortuous course of the thoracic aorta may be followed within the cardiac shadow down to the diaphragm.

In left oblique view (Plate 2) the aortic arch is seen over the left trachea and bifurcated trachea. The dilatation of the aortic arch is plainly noticeable on the left oblique view as a wide round shadow over the heart and overlapping the spine (see Plate 51).

# PLATE 31 SCLEROSIS OF THE ABDOMINAL AORTA



Anterior view Sclerosis of pelvic arteries and those of the thighs

Lateral view Marked sclerosis of the entire abdominal aorta

G S male white aged 61

Marked osteoarthritic flapping in the bodies of the lumbar vertebrae Decalcification in the bones of the pelvis and lumbar vertebrae

## PLATE 32 DISSECTING AORTIC ANEURYSM

R B male, white, aged 58 : Admitted on Dec 10, 1942, died on Dec 12 1942

Patient had been in good health had never suffered from any chronic ailment and as far as he knew had never had high blood pressure. On the day before admission he was under emotional strain. While reading a newspaper he was seized with a sudden pain in the cervical spine region. It was not excruciating, but rapidly increased in intensity and spread down the thoracic spine as low as the scapula and then radiated bilaterally about the chest. The pain was about the intensity of a toothache and seemed to throb. He was unable to sleep that night and he had an urge to defecate, associated with pain and urinary frequency every three hours. His pulse remained regular and from there was no dyspnea, palpitation, or cough.

On physical examination, he did not appear acutely ill or in great distress. Pulse 104, regular, heart and lungs negative. blood pressure 200/124. There was no tenderness along the spine. Dissecting aneurysm, diagnosed on the basis of clinical history. Shortly after admission, patient complained of blurred vision. Later he vomited but no nausea or discomfort accompanied the vomiting. On following day he felt much better. Blood pressure 165/110. Death came suddenly, within five minutes of the onset of severe dyspnea.

**Autopsy**—Chronic aortitis, tear of the aorta, dissecting aneurysm with perforation and massive left hemothorax, displacement of mediastinum to the right, compression atelectasis of the lungs.

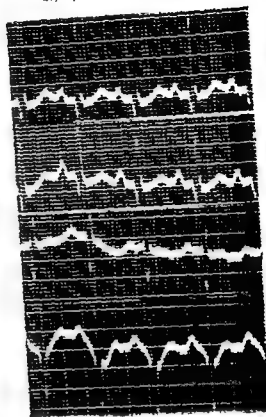
**Summary**—A case of dissecting aortic aneurysm is presented.

# PLATE 32 DISSECTING AORTIC ANEURYSM

12/10/42 Aged 59 Rate 110



1 /10/42 Prominent aortic knob



ST: s, deformed



Atherosclerosis of the aorta. Dissecting aneurysm of the thoracic aorta with perforation and massive left hemothorax. Linear transverse tear near the site of obliterated ductus arteriosus. The adventitia is dissected away from the media down to the bifurcation of the abdominal aorta and into the right common iliac artery.

## PLATE 32 DISSECTING AORTIC ANEURYSM

R B male, white aged 58 Admitted on Dec 10 1942, died on Dec 12, 1942

Patient had been in good health had never suffered from any cardiac ailment and as far as he knew had never had high blood pressure On the day before admission he was under emotional strain While reading a news paper he was seized with a sudden pain in the cervical spine region it was not excruciating, but rapidly increased in intensity and spread down the thoracic spine as low as the scapula and then radiated bilaterally about the chest The pain was about the intensity of a toothache and seemed to throb He was unable to sleep that night and he had an urge to defecate, associated with pain, and urinary frequency every three hours His pulse remained regular and firm, there was no dyspnea palpitation or cough

On physical examination, he did not appear acutely ill or in great distress Pulse, 104 regular, heart and lungs negative blood pressure 200/124 There was no tenderness along the spine Dissecting aneurysm diagnosed on the basis of clinical history Shortly after admission patient complained of blurred vision later he vomited but no nausea or discomfort accompanied the vomiting On following day he felt much better Blood pressure 165/110 Death came suddenly within five minutes of the onset of severe dyspnea

**Autopsy**—Chronic aortitis tear of the aorta dissecting aneurysm with perforation and massive left hemothorax displacement of mediastinum to the right compression atelectasis of the lungs

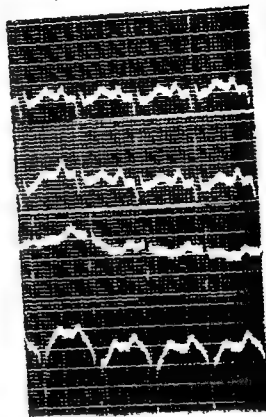
**Summary**—A case of dissecting aortic aneurysm is presented

# PLATE 32 DISSECTING AORTIC ANEURYSM

12/10/42 Aged 59 Plate 110



12/10 42 Prominent aortic knob



ST: a deformed



Atherosclerosis of the aorta Dissecting aneurysm of the thoracic aorta with perforation and massive left hemothorax Linear transverse tear near the side of obliterated ductus arteriosus The adventitia is dissected away from the media down to the bifurcation of the abdominal aorta and into the right common iliac artery

## PLATE 33 CIRROID AND ARTERIOVENOUS ANEURYSMS

I. C., male white aged 64. Admitted on March 22, 1942. died on May 11 1942

Advanced arteriosclerosis. Blood pressure, 230/160

Illustrating cirroid (varicose) aneurysm of the left common iliac artery altered by an acute red thrombosis of its lumen. This aneurysm formed proximal to an arteriovenous communication between the left femoral artery and vein (arteriovenous aneurysm) due to a stab wound in the region fifty two years previously (fig. 12)

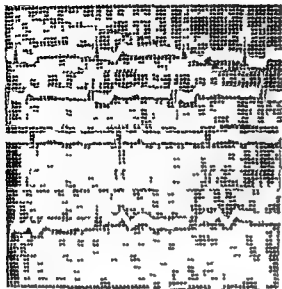
The arteriovenous aneurysm manifested itself by a continuous murmur in the region of the aneurysm due to a continuous flow of blood from the artery into the vein

Ligation of the arteriovenous aneurysm was followed by circulatory disturbance in the left lower extremity complicated three weeks later by gas gangrene infection for which amputation of the extremity below the knee was done. Death occurred five days after the amputation

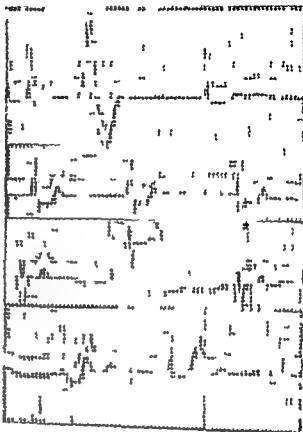
# PLATE 33 CIPSOID AND ARTERIOVENOUS ANEURYSMS

1/26/40 Aged 67

3/23/40 Aged 64 H.P. 30/160



Rate 66 Auricular fibrillation Left axis deviation



Rate 54 Auricular fibrillation Ventricular premature beats (Lead II)



Thrombosed cirroid aneurysm of the left common iliac artery formed proximal to femoral arteriovenous aneurysm due to stab wound of 22 years duration



## PLATE 34 NODULAR CALCIFYING AORTIC STENOSIS WITH CARDIAC FAILURE

C. P., male, white, aged 68 Admitted on Oct. 31, 1942, died on Dec. 1942

Complaints on admission were dyspnea on exertion of three years duration at rest for one year, ankle edema one month, tenderness in the right upper quadrant and productive cough one week. Blood pressure, 132/80. Dullness and rales over the lower lobes. The heart was greatly enlarged. There was a loud aortic systolic murmur in the third interspace to the left of the sternum. The liver extended 4½ inches below the right costal margin. There was fluid in the abdomen. No history of rheumatic fever.

Patient was getting along fairly well, when two days before death developed pulmonary infection. Pulse, 130, temperature 102.2 F, blood pressure, 140/100.

**Autopsy**—Chronic nodular calcifying aortic endocarditis stenosis, left ventricular hypertrophy. Chronic passive hyperemia of the lungs and abdominal viscera, ascites and right hydrothorax. Thrombosis of the perivascular plexus of veins, multiple small pulmonary emboli with multiple small hemorrhagic infarcts. General arteriosclerosis, chronic fibrous and calcifying pleuritis.

**Heart**—There is a marked irregular nodular rough calcification which is chiefly on the aortic surface. The stenosis is marked. Calcified nodules on the free margins and the cusps with thrombotic ulceration of one of the nodules. Agglutination of the commissures between the right and posterior cusps.

**Summary**—A case of nodular calcific aortic stenosis with cardiac failure is presented. Marked hypertrophy of the left ventricle due to aortic stenosis and marked dilatation of the left ventricle with cardiac failure correlated well with electrocardiographic findings of left ventricular strain. Death was due to pulmonary emboli and infarcts.

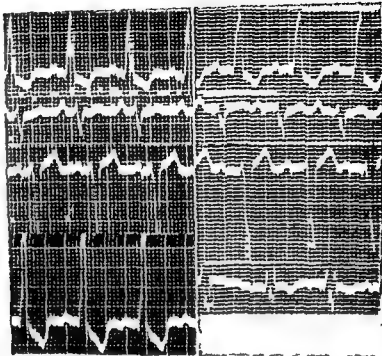
# PLATE 34 NODULAR CALCIFYING AORTIC STENOSIS WITH CARDIAC FAILURE

11/4/49 Aged 69

11/7/4 BP 1 /55



11/3/49 Heart markedly increased in all directions. Left ventricular border rounded and out. Calcific plaque left pleura. Increased lung markings.



Left axis deviation. QRS relatively high. ST<sub>1</sub> and ST<sub>2</sub> depressed and fused with inverted T<sub>1</sub>. Left ventricular strain. Similar to 11/4/42 except QRS; low notched ST<sub>1</sub>; normal T<sub>1</sub>; inverted low probably due to coronary sclerosis.



Chronic nodular calcifying aortic stenosis. Coronary sclerosis. Dilatation and hypertrophy of the left ventricle. 73 mm thick at the base.



Enlarged view of the nodular calcific aortic stenosis.

## PLATE 35    ARTERIOSCLEROSIS, AURICULAR FLUTTER AND FIBRILLATION

The abnormal auricular, "sinus" rhythm is due to abnormal initiation and transmission of auricular impulses

In auricular flutter the auricular activity is represented by flutter F,' waves large, regular, continuous deflections, which are uniform except where distorted by QRS, 1 occurring at the rate of from 200 to 380 per minute, usually recorded best in Leads 2 and 3. QRS occurs regularly at a rate which is slower than, and in a constant ratio to the auricular rate, as 2 to 1, 3 to 1, or 4 to 1. In some cases especially when treated the ratio varies in the same tracing, and ventricular rhythm is irregular. Rarely, the ratio may be 1 to 1 and such an electrocardiogram is distinguished with difficulty from those of other supraventricular tachycardias

In auricular fibrillation the auricular activity is represented by f waves small, irregular variable deflections which have usually a rate of 450 per minute. QRS occurs at irregular intervals and as some ventricular contractions are weak and do not reach the radial pulse there will be a pulse deficit

When auricular fibrillation is associated with a complete atrioventricular heart block with ventricles responding to their own intraventricular center the ventricular rhythm is regular the heart rate is slow and there is no pulse deficit

The electrocardiogram shows f waves instead of P and regular QRS of intraventricular origin with slow ventricular rate. If f' waves are not visible, absence of P and regular QRS may suggest nodal rhythm and at times differentiation may be difficult especially when the ventricular rate is slow

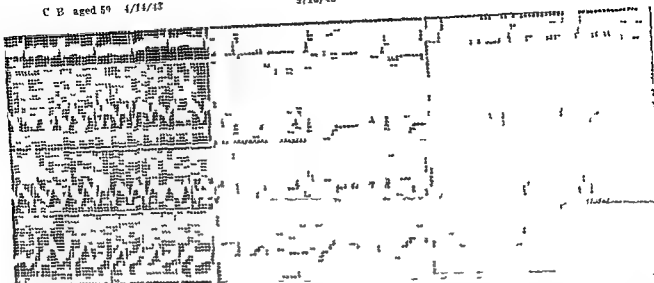
The premature systoles which are frequently encountered in auricular fibrillation are of ventricular origin as the auricles while fibrillating are unable to initiate normal or premature (auricular or nodal) contractions

# PLATE 35 ARTERIOSCLEROSIS, AURICULAR FLUTTER AND FIBRILLATION

C B aged 59 4/14/43

9/16/43

1/15/44

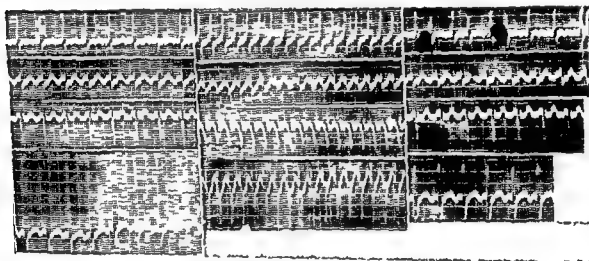


Auricular flutter of varying ratio  
(2 or 3 to 1) Ventricular rate 13

ST<sub>2</sub> : concave fused with T :  
digitalis effect Ventricular rate

Normal tracing 11/1 of previous  
month 4/14/44

F L aged 61

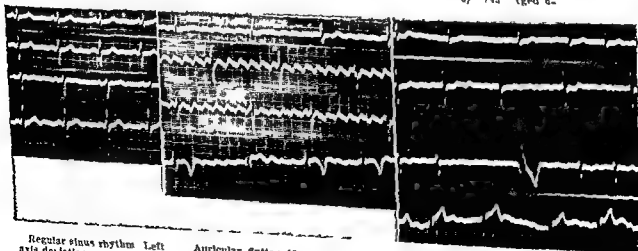


While first ICG (auricular flutter 2:1 rate of 125) was being taken patient had a  
paroxysmal attack of 1:1 flutter with rate of 150 which in a few minutes returned to  
2:1 with rate of 125 Patient died five weeks later

W R aged 55 3/16/39

9/40

6/22/43 Aged 62



Regular sinus rhythm Left  
axis deviation

Auricular flutter (6 or 5:1) QRS  
T<sub>2</sub> inverted Coronary disease

Auricular fibrillation Ventricular pre-  
mature systole QRS<sub>1</sub> inverted T<sub>2</sub> up

## PLATE 35 ARTERIOSCLEROSIS, AURICULAR FLUTTER AND FIBRILLATION

The abnormal auricular 'cactus' rhythm is due to abnormal initiation and transmission of auricular impulses

In *auricular flutter* the auricular activity is represented by flutter waves, large, regular, continuous deflections, which are uniform, except when distorted by QRS, T, occurring at the rate of from 200 to 380 per minute usually recorded best in Leads 2 and 3. QRS occurs regularly at a rate which is slower than and in a constant ratio to the auricular rate, as 2 to 3 to 1 or 4 to 1. In some cases especially when treated, the ratio varies the same time, and ventricular rhythm is irregular. Usually the ratio may be 1 to 1 and such an electrocardiogram is distinguished with difficulty from those of other supraventricular tachycardias.

In *auricular fibrillation* the auricular activity is represented by fast, small, irregular, variable deflections which have usually a rate of 450 per minute. QRS occurs at irregular intervals and as some ventricular contractions are weak and do not reach the radial pulse there will be a pulse deficit.

When auricular fibrillation is associated with a complete auriculoventricular heart block with ventricles responding to their own intraventricular center the ventricular rhythm is regular the heart rate is slow and there is no pulse deficit.

The *electrocardiogram* shows fast waves instead of P and regular QRS of intraventricular origin with slow ventricular rate. If fast waves are visible absence of P and regular QRS may suggest nodal rhythm and sometimes differentiation may be difficult especially when the ventricular rate is slow.

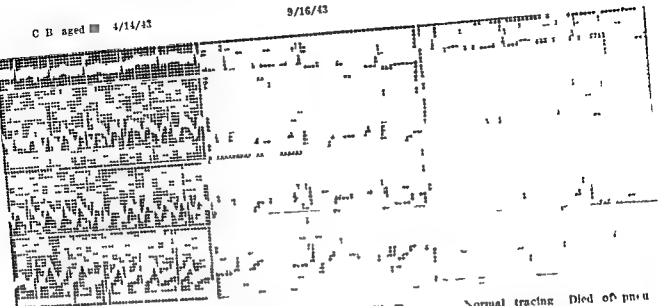
The premature systoles which are frequently encountered in auricular fibrillation are of ventricular origin as the auricles while fibrillating are unable to initiate normal or premature (auricular or nodal) contractions.

# PLATE 35 ARTERIOSCLEROSIS, AURICULAR FLUTTER AND FIBRILLATION

1°/15/43

9/16/43

C B aged 4/14/43

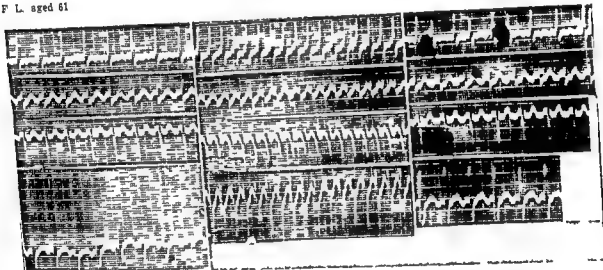


Auricular flutter of varying ratio  
(2 or 3 to 1) Ventricular rate 1°

ST = concave fused with T =  
digitalis effect Ventricular rate

Normal tracing Died of pneumonia  
month 4/14/44

F L aged 61

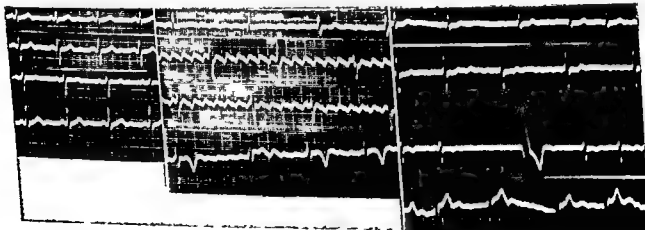


While first ECG (auricular flutter 2° rate of 1°5) was being taken patient had a  
paroxysmal attack of 1:1 flutter with rate of 200 which in a few minutes returned to  
1 with rate of 12 Patient died five weeks later

W P aged 3 15/39

9/27/4

6/ 43 16 d 6



Regular sinus rhythm Left  
axis deviation

Auricular flutter (6 or 1) QRS,  
T inverted Coronary disease

Auricular fibrillation Ventricular prema  
ture systole QRS inverted T, up

## PLATE 35    ARTERIOSCLEROSIS, AURICULAR FLUTTER AND FIBRILLATION

The abnormal auricular "circuit" rhythm is due to abnormal initiation and transmission of auricular impulses

In **auricular flutter** the auricular activity is represented by flutter "F," waves, large, regular, continuous deflections which are uniform, except where distorted by QRS, 2, occurring at the rate of from 200 to 380 per minute usually recorded best in Leads 2 and 3. QRS occurs regularly at a rate which is slower than and in a constant ratio to the auricular rate as 2 to 1, 3 to 1, or 4 to 1. In some cases especially when treated the ratio varies in the same tracing, and ventricular rhythm is irregular. Rarely, the ratio may be 1 to 1 and such an electrocardiogram is distinguished with difficulty from those of other supraventricular tachycardias.

In **auricular fibrillation** the auricular activity is represented by "f" waves small, irregular, variable deflections which have usually a rate of 450 per minute. QRS occurs at irregular intervals and as some ventricular contractions are weak and do not reach the radial pulse there will be a pulse deficit.

When auricular fibrillation is associated with a complete auriculoventricular heart block with ventricles responding to their own intraventricular center the ventricular rhythm is regular the heart rate is slow and there is no pulse deficit.

The electrocardiogram shows f waves instead of P and regular QRS of intraventricular origin with slow ventricular rate. If "f" waves are not visible absence of P and regular QRS may suggest nodal rhythm and at times differentiation may be difficult, especially when the ventricular rate is slow.

The premature systoles which are frequently encountered in auricular fibrillation are of ventricular origin as the auricles while fibrillating are unable to initiate normal or premature (auricular or nodal) contractions.

disappear as soon as the redness and swelling in the area surrounding the myocardial infarct will subside

In myocardial infarct, especially when extensive, the balance of the left ventricular myocardium may fail, the myocardial insufficiency in this case being the result of a marked coronary insufficiency. Clinically, there will be dyspnea as a sign of left ventricular failure. The electrocardiogram will show tachycardia, marked left axis deviation, low voltage of all the leads (QRS-T), or the ST-segment in leads 1 and 4 or 2 and 3 instead of high take off or elevation may be markedly depressed, concave and filled with T's.

In some cases, the myocardial infarct may be the focus for arrhythmic irritability and cause appearance of premature systoles or paroxysmal ventricular tachycardia, flutter or fibrillation.

The localized necrosis in myocardium leads to a localized involvement of endocardium with formation of a mural thrombus and of pericardium with pericarditis. The thrombus may lead to emboli in systemic circulation and if a vital organ, as the brain is involved, may be the direct cause of death, while the myocardial infarction itself was clinically improving. The localized pericarditis may cause a pericardial rub, heard usually for a short time anteriorly, in anterior infarct. When the pericarditis is extensive not localized, the pericardial rub may be present over a long period of time, and also may be heard in posterior infarct.

The necrotic myocardium may rupture. If the wall ruptures, the hemorrhage may cause heart tamponade and death.

The rupture of the interventricular septum may heal without any ill effect except for appearance of a systolic murmur in the region of the third or fourth left interspace near the sternal border. While the congenital interventricular septum defect is in the lower, membranous upper portion of the septum, the acquired septum defect is in the muscular portion. In congenital septum defect, if associated with pulmonary stenosis and increased pressure in the right ventricle, the venous blood of the right ventricle flows into the left ventricle, mixes with arterial blood and causes central cyanosis. In acquired septum



## CORONARY SCLEROSIS AND MYOCARDIAL INFARCTION

Atherosclerosis of the intima of the coronary arteries is a process similar to that in any other arteries, but the changes resulting from it are of great importance as to the proper function of the myocardium.

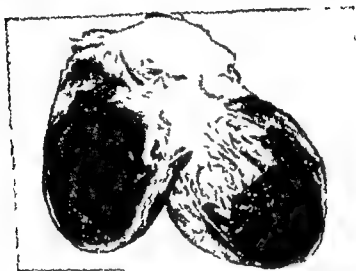
The coronary sclerosis may be silent and inactive with slight changes in QRS, ST, and T as the only evidence, if conduction in ventricular myocardium is impaired, or various types of heart block, incomplete, complete, or bundle branch block may be present, if the septum is involved.

The coronary sclerosis may be active. Clinically it will manifest itself by dull or sharp precordial pain on exertion or angina pectoris as due to insufficient blood supply and myocardial anoxemia, or dyspnea on exertion due to myocardial insufficiency and left ventricular failure. In the electrocardiogram there will be noted changes in various abnormalities found before as inverted QRS, T's or ST changes when compared with previous electrocardiograms, those comparative changes are electrocardiographic signs of activity of coronary sclerosis.

Atherosclerosis of the intima of the coronary arteries may lead to thrombosis and occlusion of the lumen of a branch of a coronary artery, and thus in turn to a localized myocardial infarction and necrosis. This is most frequently encountered in two locations (1) in the apex, anterior part of the left ventricle and adjoining portion of the interventricular septum, so called anterior infarct, a part supplied by anterior descending branch of the left coronary artery and, (2) in the base, posterior part of the left ventricle and adjoining posterior portion of the interventricular septum, so called posterior infarct a part supplied by the circumflex branch of the left coronary or by the right coronary artery.

In early stage of infarction, the electrocardiogram shows marked changes in QRS, ST, and T, especially elevation and high take off of ST in Leads 1 and 4 in anterior, and Leads 2 and 3 in posterior infarcts. Quite often, the myocardium in the vicinity of the necrotic area may also be involved, being red and swollen and increasing the interference with myocardial conduction. Therefore, it is not unusual to find in the first day or two of infarction extensive electrocardiographic changes, including various degrees of heart block, which will

**PLATE 36 RECENT AND OLD ANTERIOR MYOCARDIAL  
INFARCTS WITH LARGE MURAL THROMBUS**



**P. M. white male aged 61**

Acute coronary thrombosis a year ago advanced cardiac failure in the last three months. Recent (silent) anterior infarct with a large organizing mural thrombus superimposed upon an (one year) old healed anterior infarct with ventricular aneurysm. Marked dilatation of the left ventricle advanced cardiac failure, chronic passive hyperemia of the lungs liver spleen and other viscera bilateral hydrothorax hydropericardium ascites and edema of right ankle. Recent thrombosis of the periprostatic veins. Large recent pulmonary infarct.

defect, the same is in congenital without pulmonary stenosis the pressure in the left ventricle remains higher than in the right, the arterial blood from the left ventricle flows into the right, and there will be no central cyanosis

The acute myocardial infarction may heal without leaving any clinical or electrocardiographic evidence of impairment. In others, the infarcted area may cause localized thinning and bulging of the wall, a ventricular aneurysm, and impairment in myocardial conduction, with permanent changes in QRS, ST or T as residue of a healed infarct, and clinically dyspnea as a sign of left ventricular insufficiency and cardiac failure, which, if unchecked, may gradually progress to advanced cardiac failure and death. In advanced cardiac failure it is not unusual to find a thrombosis in some peripheral vein causing pulmonary embolism with infarction and death.

In conclusion it is worth mentioning that one episode of myocardial infarction may be followed later by another in the same or another area, with the consequences already stated.

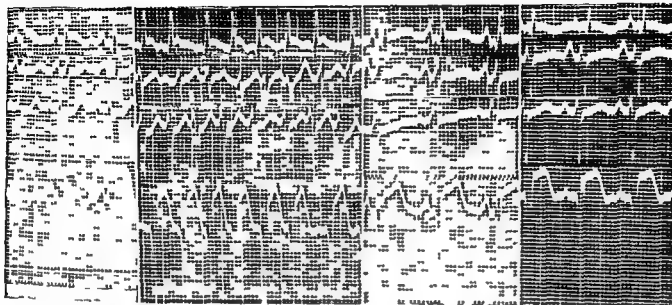
# PLATE 37 RECENT ANTERIOR INFARCT WITH PAROXYSMAL AURICULAR FIBRILLATION

1° 9/39

1° 29/39

1° 30/39

2° 14/40



Rate 110  
Left axis deviation  
Q present ST high take  
off and fused with T

Rate 10  
Paroxysmal auricular fibrillation

Rate 110  
Regular sinus rhythm  
high peaked  
Auricular hypertrophy

Rate 115  
QRS, low ST, near nor  
mal T; low inverted

QRS, inverted ST markedly elevated and fused with T

Very slight ECG changes in QRS ST and T over  
period of six weeks



2/40 Left ventricular hypertrophy Increased  
markings—cardiac

Organizing myocardial infarct of the anterior wall of  
the left ventricle and septum with mural thrombus

## PLATE 37 RECENT ANTERIOR INFARCT WITH PAROXYSMAL AURICULAR FIBRILLATION

W R, male, white, aged 57 Admitted on Dec 26, 1939, died on Feb 15, 1940

Hypertension known to be present since June, 1937 The chief complaint on admission was constant, severe pain under the lower part of the sternum of a few hours' duration, unaffected by bed rest and only slightly relieved by morphine The pain radiated toward the right shoulder and arm There was constant nausea and the patient vomited a greenish liquid a few times at home A severe dyspnea set in shortly after the pain began On admission, patient was breathing with difficulty, the general color was good the lips and hands were only moderately cyanotic Heart moderately enlarged to left, the rate was regular the sounds of good quality no murmurs Blood pressure 172/106; Leucocyte count 16,650 Temperature reached the peak of 102° F two days after admission and remained between 98.6 and 100° F for the rest of the course in the hospital The pulse varied from 100 to 130 respirations from 20 to 35

On Dec 28 1939, there was an arrhythmia and friction rub heard under the sternum Next day the pain disappeared there was pulse deficit a continued substernal friction rub was heard Nine days after admission the dyspnea increased in severity The condition was unchanged until Feb 14 1940 when the patient suddenly expired

**Autopsy**—Extensive organizing infarct of the anterior wall of the left ventricle and ventricular septum Extensive mural thrombus of the left ventricle Marked sclerosis and recanalized lumen of the descending branch of the left coronary artery Generalized arteriosclerosis Bilateral hydrothorax with slight compression atelectasis of the lower lobes Cyanotic in duration of the lungs

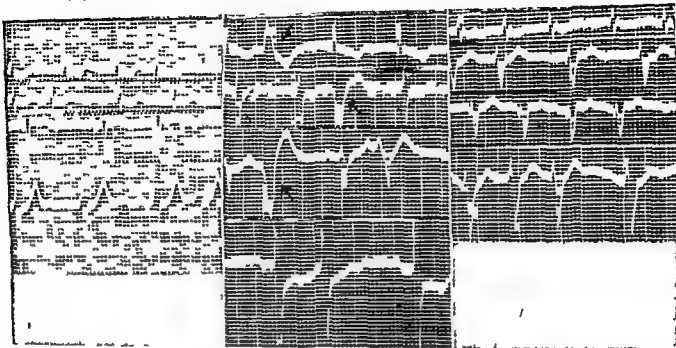
**Summary**—A case of recent anterior myocardial infarct with typical electrocardiographic findings in Leads 1 and 4 is presented Paroxysmal auricular fibrillation appeared two days after admission and disappeared two days later There were very slight electrocardiographic changes noted over a period of six weeks and the patient died of left ventricular failure due to extensive myocardial infarction

# PLATE 38 RECENT ANTERIOR MYOCARDIAL INFARCT WITH CARDIAC FAILURE

5/15/43 Aged 70

6/7/43 BP 170/100

6/15/43 Rate 119



T diphasic

Ventricular premature systoles T<sub>i</sub> deeply inverted coved ST<sub>i</sub> depressed T<sub>i</sub> isoelectric

QRS<sub>i</sub> low QRS<sub>i</sub> inverted T<sub>i</sub> | o electric T<sub>i</sub> low

Auricular fibrillation and left axis deviation present in all three tracings. Changes in Leads I and 4 (QRS ST T) on various dates indicate recent anterior myocardial infarct. Auricular fibrillation was present before the infarction.



Left ventricle opened from the front showing aorta and aortic valve. Mitral valve to the right. Large recent organizing infarct apex of the left ventricle.



Left ventricle opened from the back showing left auricle mitral valve. Mural thrombus of left ventricle and left auricular appendage.

The wall of the left ventricle in infarcted area is only 3 mm thick at the base 6 mm left ventricular hypertrophy due to associated hypertension (left axis deviation in electrocardiogram).

## PLATE 38 RECENT ANTERIOR MYOCARDIAL INFARCT WITH CARDIAC FAILURE

J C, male, white, aged 70. Admitted on June 8, 1943, died on June 21, 1943

Patient had been in good health for about three months prior to admission when he began to have cramping precordial pain radiating down to left arm, on exertion or emotion lasting but a few minutes. The pains were ascribed by his physician to coronary disease. At 1 A M on the day of admission he was awakened by a severe pain, not relieved by morphine. He was admitted early in the morning, however, without much pain. On admission pulse, 100 temperature, 100 F blood pressure 170/100. Left cardiac border 2 cm behind the midclavicular line. auricular fibrillation. Good cardiac sounds with a precordial systolic murmur. Leucocyte count 20 000

His condition was characterized by intermittent periods of pulmonary congestion mental confusion cyanosis and dyspnea

**Autopsy**—Extensive arteriosclerosis of the coronary arteries and aorta thrombosis of anterior descending branch of left coronary artery large recent organizing infarct apex of the left ventricle large mural thrombus of the left ventricle and left auricular appendage Hypertrophy of cardiac chambers especially the left ventricle the wall of the left ventricle in the region of the infarct is not over 8 to 10 millimeters in thickness in contrast to the basal portion which reaches a maximum of 20 millimeters

Thrombosis of periprostatic veins bilateral pulmonary embolism thrombosis with hemorrhagic infarcts of right lower lobe hyperemia and edema of lungs bilateral hydrothorax (right 500 cc and left 200 cc) Chronic passive hyperemia of the lungs liver spleen and kidneys

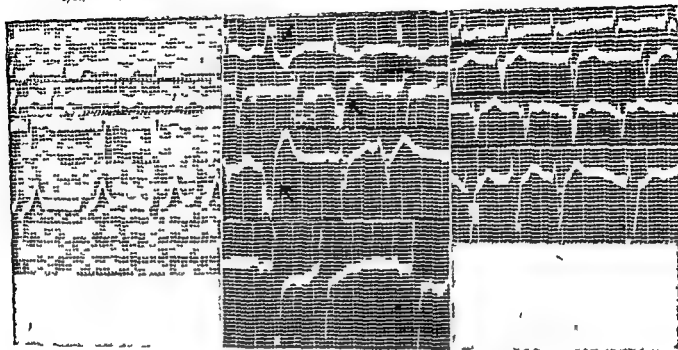
**Summary**—A case of recent anterior myocardial infarct is presented with hypertension and auricular fibrillation present prior to infarction. Death due to advanced cardiac failure and pulmonary embolism thrombosis with pulmonary infarcts probably from thrombosis of periprostatic veins

# **PLATE 38 RECENT ANTERIOR MYOCARDIAL INFARCT WITH CARDIAC FAILURE**

5/15/43 Aged 70

6/7/43 BP 170/100

6/15/43 Rate 116



T diphasic

Ventricular premature systoles T<sub>i</sub>  
deeply inverted cored ST depressed  
T isoelectric

QRS<sub>1</sub> low QRS<sub>2</sub> inverted T<sub>1</sub> i o-  
electric T low

Auricular fibrillation and left axis deviation present in all three tracings. Changes in Leads 1 and 4 (QRS ST T) on various dates indicate recent anterior myocardial infarct. Auricular fibrillation was present before the infarction.



Left ventricle is opened from the front showing aorta and aortic valve. Mitral valve to the right. Large recent organizing infarct apex of the left ventricle.



Left ventricle opened from the back showing left auricle, mitral valve, mural thrombus of left ventricle and left auricular appendage.

The wall of the left ventricle in infarcted area is only 8 mm thick at the base (11 mm left ventricular hypertrophy due to associated hypertension (left axis deviation in electrocardiogram)).



## PLATE 39 OLD CALCIFIED ANTERIOR INFARCT WITH VENTRICULAR ANEURYSM

A. B., male, white, aged 66. Admitted on March 17, 1942, died on March 21, 1942.

Patient had a coronary attack thirteen years prior to admission at 53 years of age, and remained in bed for a month. He weighed 210 pounds at the time but gradually reduced to 165 pounds. Ten years later he developed diabetes mellitus. A year before admission he began to have attacks of nocturnal dyspnea as a sign of advanced left ventricular failure, but no precordial pain. The examination made at that time revealed heart regular rhythm, no murmurs, liver enlarged with fluid in abdomen, edema of lower extremities. Blood pressure 140/70. Urine: albumin 1 to 3 plus, no sugar, occasional red cells, a few hyaline granular casts. He felt well until his last admission with acute bronchopneumonia (March 17, 1942) he died four days later.

**Autopsy**—Old healed occlusion of the anterior descending branch of the left coronary artery; old, healed infarction with parietal aneurysm, mural thrombosis and calcification of the wall of the left ventricle. Chronic dilatation and disseminated calcification of the mitral ring. Chronic passive hyperemia of the lungs, liver (congestive induration), spleen and kidneys. Acute bronchopneumonia. Fat infiltration of the pancreas (clinical diagnosis of diabetes mellitus). Slight benign nephrosclerosis.

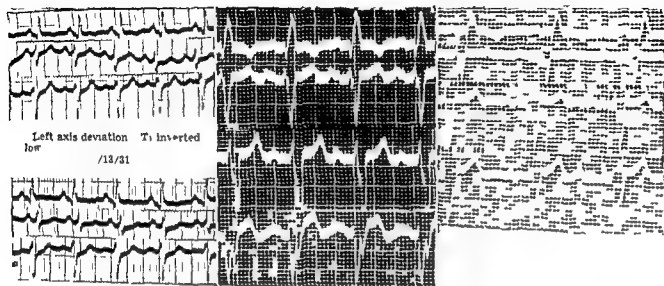
**Summary**—A case of an old anterior myocardial infarct with a large ventricular aneurysm is presented. The large portion of the wall of the aneurysm consisted of bone and calcified scar tissue. This change developed over a period of thirteen years. Death was due to intercurrent bronchopneumonia.

# PLATE 39 OLD CALCIFIED ANTERIOR INFARCT WITH VENTRICULAR ANEURYSM

2/2/31 Aged 55

4/ 3/41 Aged 65

1/29/47 Aged 67



The persistent changes in Lead 1 (Q<sub>1</sub> present T inverted) and Lead 4 (QR<sub>4</sub>, in-  
verted ST<sub>4</sub> slightly elevated and deformed) suggest an old healed anterior infarct  
with ventricular aneurysm. The persistent left bundle branch block suggests involve-  
ment of the interventricular septum.



## PLATE 40 DISSECTING ANEURYSM OF THE LEFT CORONARY ARTERY OLD ANTERIOR INFARCT WITH PERFORATED SEPTUM

P B, male, white aged 71 Admitted on June 2, 1942, died on June 29, 1942

Patient had been in good health when, on the day of admission, he was awakened with severe precordial and epigastric pain, which radiated bilaterally to the back and scapular regions and down both arms to the elbows he broke out into a cold sweat The pain continued without abating for several hours and in the morning, he vomited some food He had never had any pains previously He had had hypertension (over 200) for a few years

The heart was enlarged to the left and right the tones were distant no murmurs blood pressure, 138/94 Abdomen distended White count 19 000 On the eighth day the patient developed pulmonary infarction and ascites He died suddenly on the twenty eighth day

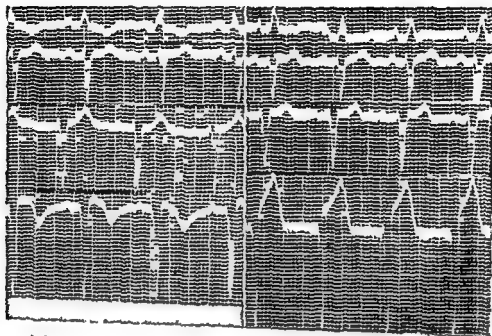
**Autopsy**—Generalized and marked coronary arteriosclerosis but no thrombi Dissecting aneurysm of the anterior descending branch of left coronary artery, causing its obstruction Old healed infarct of the anterior wall of the left ventricle and ventricular septum with parietal aneurysm The interventricular septum is exceptionally thin and there is a parietal aneurysm of the septum bulging from left to right side In the central portion there is an ovoid septal defect the edges are thickened smooth firm pearly white suggesting that they are made of thickened tissue and that defect has been present for a long time Apical mural thrombus of left ventricle Dilatation and hypertrophy (20 mm) of the left ventricle Acute hemorrhagic (aspiration) bronchopneumonia Pulmonary embolic thromboses with hemorrhagic infarct of right lower lobe Bilateral hydrothorax ascites and general anasarca due to cardiac failure

**Summary**—A case of dissecting aneurysm of descending branch of left coronary is presented superimposed upon an asymptomatic old healed anterior infarct with aneurysm and perforated interventricular septum Death due to cardiac failure bronchopneumonia and pulmonary embolism thromboses with infarct

**PLATE 40 DISSECTING ANEURYSM OF THE LEFT CORONARY  
ARTERY OLD ANTERIOR INFARCT WITH PERFORATED  
SEPTUM**

6/3/49 Aged 41

6/9/49



Left axis deviation QRS low QRS,  
inverted T<sub>1</sub> & inverted coved

T<sub>1</sub> low, inverted ST<sub>1</sub> markedly  
elevated deformed fused with T<sub>1</sub>

The changes noted in Leads I and 4 especially in ST<sub>1</sub> and T<sub>1</sub> indicate active recent  
anterior coronary disease



6 0 4 Heart enlarged to the left and right as in  
cardiac failure with dilatation of both ventricles. Bron-  
chopneumonia



Old healed anterior myocardial infarct. Ventricular  
aneurysm Old healed perforated interventricular  
septum defect

## PLATE 41 RECENT POSTERIOR INFARCT WITH ACUTE PERICARDITIS

P. R., male, white, aged 63. Admitted on Jan. 2, 1942, died on Jan. 1, 1942.

Two and one half months previous to admission the patient had an attack of precordial pain, accompanied by dyspnea. Five weeks later, the patient developed a substernal pain which was different than he had experienced previously, the pain did not radiate to the left arm or neck, and was not related to food taken or exercise.

On admission pulse 86, temperature,  $101.8^{\circ} \text{F}$ , blood pressure, 128/78. The heart sounds were distinct, regular, no murmurs. The liver was two fingerbreadths below the costal margin, slight pitting, and leg edema. White count, 21,600. Four days later there was a definite tenderness over the lower precordium. One day before death, patient complained of pain in precordium and left shoulder, he was apprehensive, his color was ashen, there were rales in posterior base of the lungs, pulse 130 to 145. Blood pressure 130/105. Patient died next morning.

**Autopsy**—Atherosclerosis of the aorta and coronary arteries with calcification and ulceration. Recent occlusion of the circumflex branch of the left coronary artery organizing recent myocardial infarction, posterior wall of the left ventricle near base organizing, acute sanguinoserosofibrinous pericarditis, hypertrophy and dilatation of the left and right ventricles and dilatation of right auricle. Chronic passive hyperemia of the lungs, liver, spleen and kidneys. Moderate bilateral hydrothorax, compression atelectasis of both lower pulmonary lobes. Organizing infarct of the spleen, arteriosclerotic atrophy of the kidneys.

**Summary**—A case of a recent posterior myocardial infarct with acute pericarditis and cardiac failure is presented. Although the infarct was posterior a pericardial rub was heard anteriorly due to generalized pericarditis.

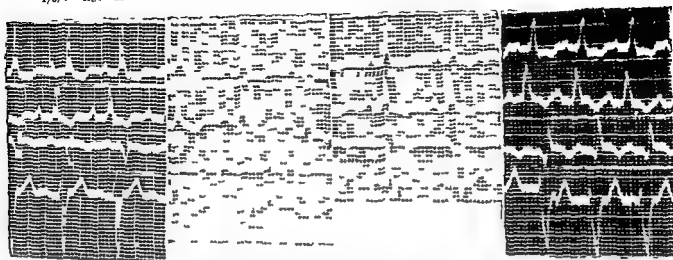
# PLATE 41 RECENT POSTERIOR INFARCT WITH ACUTE PERICARDITIS

1/3/4 Aged ■

1/7/4°

1/8/4°

1/13/4°



The electrocardiographic changes in Leads 2 and 3 (Q; present, high take-off of ST; T is inverted) suggest a recent posterior infarct in addition to active anterior coronary disease (T; low biphasic or inverted QRS; inverted or low)



Heart markedly enlarged in all directions due to pericarditis and cardiac failure

Recent posterior myocardial infarct acute non-thrombotic pericarditis

## PLATE 42 OLD POSTERIOR INFARCT WITH VENTRICULAR ANEURYSM

I B male, white aged 61 Admitted on March 14, 1935, died on May 5, 1935

The patient had been in ill health periodically for twelve years Nine years before admission he had been told that he had angina pectoris In the last six years, he had had dyspnea, which was his chief complaint on admission On physical examination the heart was enlarged to the left the rate regular, no murmurs there was cyanosis of the lips, pulsation of the veins of the neck, dyspnea Blood pressure 190/120 After bed rest, blood pressure fell to 130/70

On further questioning, patient stated that in the past four years he had begun to manifest some signs of cerebral vascular disturbance with mental instability, most marked in the past year and with periods of irrationality and excitement The patient died quite suddenly on May 5, while talking to the attending physician The death was ascribed to a cerebral accident

**Autopsy**—Marked generalized arteriosclerosis, arteriosclerotic occlusion of the right coronary artery Old myocardial infarct and parietal aneurysm of the posterior wall of the left ventricle and interventricular septum localized fibrous pericarditis mural thrombosis hypertrophy of both cardiac ventricles Old dissecting aneurysm of the right common iliac artery

Arteriosclerotic atrophy of the kidneys healing infarcts of the kidneys  
*Acute distention of the lungs*

No permit for autopsy of the brain could be obtained

**Summary**—A case of an old posterior myocardial infarct with a ventricular aneurysm is presented Death was probably due to a cerebral accident

# PLATE 42 OLD POSTERIOR INFARCT WITH VENTRICULAR ANEURYSM

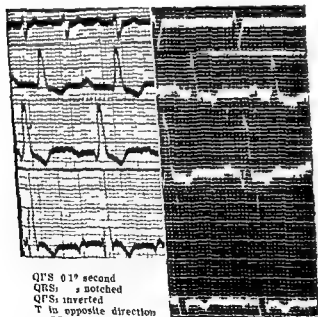


9/15/35 Left ventricular hypertrophy due to hyper-  
tension. Left border is rounded and out

3/15/35 Marked left ventricular hypertrophy and  
dilatation.

3/30/35 A=ed 61

4/10/35 BP 190/120



QRS 0.19 second  
QRS: notched  
QRS: inverted  
T in opposite direction  
to QRS

Similar to 3/30/35

Right bundle branch block. ST depressed de-  
formed T; inverted. Posterior myocardial infarct  
of the base of left ventricle with septum involvement  
(left bundle branch block)



Old posterior infarct with ventricular aneurysm of  
posterior wall of the left ventricle base and septum



## PLATE 43 RECENT ANTERIOR AND POSTERIOR INFARCTS

C W, white, female, aged 64 Admitted on April 20, 1939 died on April 28 1939

History of hypertension (200 to 220) in previous years with several attacks of epileptiform convulsions due to cerebral arteriosclerosis Nine months before admission there was a hacking cough which persisted for five months The roentgenogram of the chest revealed a markedly dilated heart Patient was relatively well until nine days before admission when she had a coughing spell with marked perspiration coolness and pallor with blood pressure below 100

On admission marked dyspnea cough cyanosis of lips and hands Pulse 70, temperature 99.4 F, respirations, 26 blood pressure 164/90 The apex is diffuse, in anterior axillary line premature systoles, apical systolic murmur Right pleural effusion Liver not enlarged no ascites no ankle edema

Her condition became serious five days after admission because of marked dyspnea, cyanosis, and stupor temperature rose to 102.4° F

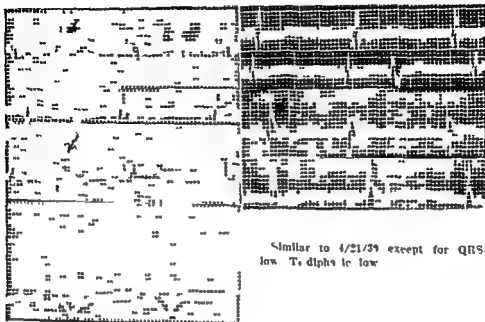
**Autopsy**—Generalized arteriosclerosis marked sclerosis of coronary arteries, recent thrombosis of the descending branch of left coronary and recent dissecting aneurysm of right coronary artery recent infarction of the anterior and posterior walls of the left ventricle and ventricular septum organizing mural thrombi of the left ventricle and auricle dilatation of both ventricles and auricles hypertrophy of right ventricle Numerous organizing thrombi of the pulmonary artery hemorrhagic infarcts of the lungs right hydrothorax chronic compression atelectasis of right lung bronchiectasis sclerosis of the pulmonary arteries

**Summary**—A case of recent anterior and posterior infarcts discovered at autopsy is presented Patient was admitted with a chronic respiratory infection and died of pulmonary infarcts

# PLATE 43 : RECENT ANTERIOR AND POSTERIOR INFARCTS

4/21/39 Aged 61

4/21/39 HP 164/90



Similar to 4/21/39 except for QRS;  
low T<sub>2</sub> diphasic low

Regular rhythm except for occasional ventricular premature systole and absent I suggest nodal rhythm or auricular fibrillation with complete auriculoventricular heart block Low voltage of all limb leads (QRS T) suggests myocardial (coronary) insufficiency



Heart shadow increased in all directions as in cardiac failure  
Right hydrothorax

Recent anterior and posterior infarcts  
Large thrombus in the left auricle

## PLATE 44 RECENT ANTERIOR AND OLD POSTERIOR INFARCTS WITH CARDIAC FAILURE

H S male, white aged 51 Admitted on Nov 23 1941 died on Dec 4 1941

Patient was well until eighteen months before admission at which time he began having pains in the right upper quadrant with tiredness and weakness. He grew progressively worse and developed dyspnea on exertion and edema of the ankles. Eight months before admission he was put on digitalis; he felt better and was able to carry on light work for two months; then he suffered a relapse with dyspnea on slightest activity with fluid in abdomen and ankle edema. He gradually improved and felt well until six weeks before admission when the edema, dyspnea and weakness returned.

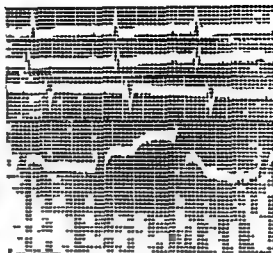
On admission pulse 90 temperature 98.4° F respirations 24 blood pressure 120/80. Heart regular rhythm no murmurs; the liver was palpated two fingerbreadths below the right costal margin with some tenderness; fluid in abdomen; edema of the lower extremities; no dyspnea while in bed; no cyanosis. He grew gradually worse with a steady increase in temperature, pulse, respiration and cyanosis. He became irrational and delirious.

**Autopsy**—Recent occlusion of the anterior descending branch of the left coronary artery with an organizing infarct of the anterior portion of the ventricular septum and left ventricle with a mural thrombus. Old healed occlusion of the circumflex branch of the left coronary with a healed infarct of the posterior portion of the left ventricle and ventricular septum at the base, with a parietal aneurysm. Dilatation of the left ventricle; hypertrophy of the right ventricle; chronic dilatation of the right heart chambers. Marked bilateral hydrothorax; ascites; chronic passive hyperemia of abdominal viscera; acute bronchopneumonia. Emboli thromboses of branches of the pulmonary artery to the right upper lobe with hemorrhagic infarct.

**Summary**—A case of recent anterior and old posterior infarcts with advanced heart failure is presented. Death due to pulmonary infarct.

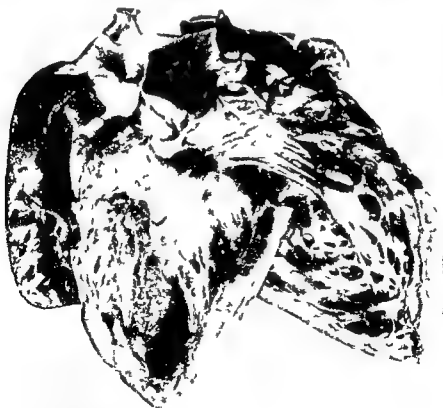
# PLATE 44 RECENT ANTERIOR AND OLD POSTERIOR INFARCTS WITH CARDIAC FAILURE

11/25/41 Aged 51 Rate 14



Left axis deviation Leads 1 2 3 (QRS T)  
of low voltage myocardial insufficiency QRS,  
inverted T, low diphasic coronary disease  
Prolonged PR—0.24 second Incomplete auricu-  
lar block suggests ventricular septum  
involvement

11/23/41 Heart markedly enlarged as in advanced cardiac failure  
Bilateral bronchopneumonia



Recent anterior infarct with mural thrombus Old healed posterior infarct  
with ventricular aneurysm Dilated left ventricle (cardiac failure)

## PLATE 45 ANTERIOR MYOCARDIAL INFARCT

The infarct is in the anterior wall, apex of the left ventricle, and anterior portion of the interventricular septum. It results usually from occlusion of the descending branch of the left coronary artery, supplying the above region.

In acute anterior infarction, the electrocardiographic changes occur in Lead 1, Q<sub>1</sub>T<sub>1</sub> pattern QRS<sub>1</sub> low voltage, Q<sub>1</sub> prominent (while S<sub>1</sub> reciprocally deep) ST<sub>1</sub> elevated high take off, and fused with T<sub>1</sub> (while ST<sub>3</sub> reciprocally depressed and fused with T<sub>3</sub>). Leads 2 and 4 usually show changes similar to Lead 1, as Lead 4 is the apical precordial lead and in close proximity to the infarcted area.

It may take a few hours to a day for the above changes to appear in an acute coronary attack, and it is not unusual to find a normal electrocardiogram (normal conduction) in the first few hours of an acute attack.

In a few days while myocardial infarct shows signs of healing myocardial conduction improves and some of the electrocardiographic changes gradually return to normal. ST returns to base line but T<sub>1</sub> & become deeply inverted, coved and QRS<sub>1</sub> inverted (normally diphasic).

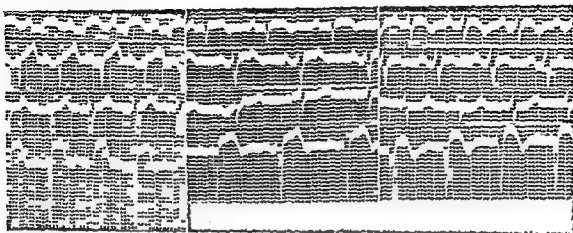
The electrocardiographic changes of healing myocardial infarct (inverted T) may persist for a few weeks and gradually return to normal in which case a small myocardial scar (fibrosis) remains as the only result of a healed myocardial infarct. If the scar (fibrosis) is extensive or as frequently happens a ventricular aneurysm develops then the ventricular conduction is permanently impaired and the electrocardiographic changes of a healing myocardial infarct with inverted T remain for years. If the electrocardiograms taken at various dates over a long period of time show persistent changes a ventricular aneurysm should be strongly suspected. This condition often causes a myocardial (coronary) insufficiency with left ventricular failure and dyspnea on exertion.

# PLATE 45 ANTERIOR MYOCARDIAL INFARCT WITH CHANGES IN LEADS 1 AND 4

3 14/40 Aged 48

3/21/45

7/1/40

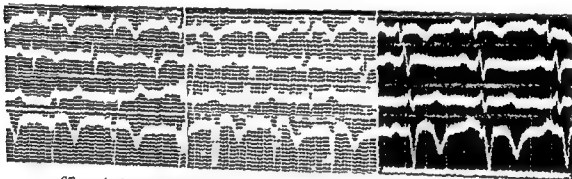


Left axis deviation suggests left ventricular hypertrophy. QRS low, inverted QRS, inverted ST, elevated fused with T, Above T-C changes in Leads 1 & 4 suggest active impairment in myocardial conduction due to acute infarction in anterior wall apex of the left ventricle.

4/9/4

4 16/4

5/1/40

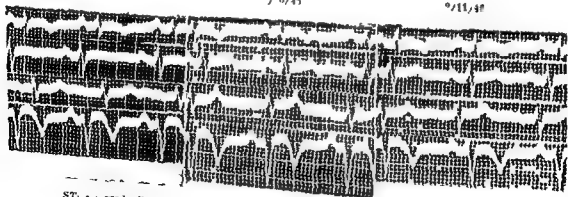


ST back to base line and convex now. T<sub>1</sub> inverted covered. Above T-C changes suggest improvement in myocardial conduction and healing of the infarction.

7/40

10/40

9/11/40



ST<sub>1</sub> gradually became a normal base line. T<sub>1</sub> low, inverted not covered. T<sub>1</sub> low, inverted not covered. Above changes over period of eleven months suggest a gradual improvement in myocardial conduction. If inversion of QRS and T remain permanently unchanged, development of ventricular aneurysm in an old healed infarcted area is to be considered. Electrocardiogram taken on 10/1/46 is similar to 11/46. No changes.

## PLATE 46 POSTERIOR MYOCARDIAL INFARCT

The infarct is in the posterior wall, base of the left ventricle and posterior portion of the interventricular septum. It usually results from occlusion of the circumflex branch of the left coronary or of the right coronary artery, supplying the above named region.

In acute posterior infarction the electrocardiographic changes occur in Leads 2 and 3, QsTs pattern. QRS low Q is prominent (while Q<sub>1</sub> absent) STs elevated and fused with T<sub>s</sub> (while ST<sub>1</sub> reciprocally depressed and fused with R<sub>1</sub>).

In a few days while myocardial infarct shows signs of healing, the myocardial conduction improves and some of the electrocardiographic changes gradually return to normal. ST returns to base line but T<sub>s</sub> become deeply inverted.

The electrocardiographic changes of healing infarct (inverted T<sub>s</sub>) may persist for a few weeks and gradually return to normal, or remain as such unchanged for months or years suggesting a ventricular aneurysm.

### Case 1 M C female white aged 71

#### Acute posterior infarct with rupture of the heart

Hypertension for fifteen years. In the last year intermittent attacks of vicelike substernal pain on exertion or occasionally after eating radiating down both arms relieved by rest—coronary disease. The evening before admission acute attack of sharp precordial pain nausea and vomiting no dyspnea. Blood pressure 206/100. Heart regular no murmurs. Fever 99 to 102.4° F. Leucocyte count 13 600. On the fourth day the blood pressure was 168/88 and patient died rather suddenly.

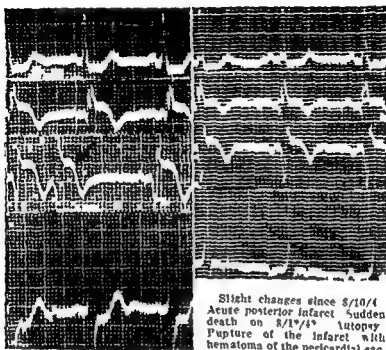
**Autopsy**—Marked aortic and coronary arteriosclerosis. Acute occlusion of the posterior descending branch of the right coronary artery due to hemorrhage into an arteriosclerotic plaque. Acute infarction of the posterior wall of the left ventricle. Spontaneous rupture of the infarct with hematoma of the pericardial sac.

# PLATE 46 POSTERIOR MYOCARDIAL INFARCT WITH CHANGES IN LEADS 2 AND 3

CASE 1

M C aged 1 8/10/4

9/10/4



Slight changes since 8/10/4  
Acute posterior infarct Sudden  
death on 8/17/49 Autopsy  
Rupture of the infarct with  
hematoma of the pericardial sac

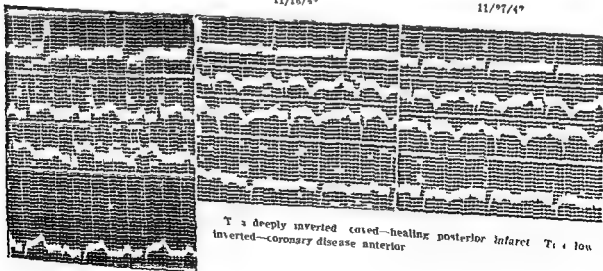
Q prominent STs + high take-  
off T + inverted Premature sys-  
tole (Lead 3)

CASE 2

A P aged 10/30/49

11/16/49

11/27/49



T + deeply inverted coved—healing posterior infarct T<sub>1</sub> + low  
inverted—coronary disease anterior

ST high take-off fused with  
T<sub>1</sub>

Two weeks after admission patient develop d acute thrombophlebitis of left femoral  
vein followed later by pulmonary embolism and death on 1/3/50

Autopsy Death due not to the posterior myocardial infarct but to pulmonary  
embolism from thrombosis of the l R femoral vein



## PLATE 47 MYOCARDIAL INFARCTION WITH PAROXYSMAL AURICULAR FLUTTER

In myocardial infarct, especially when extensive, the unaffected portion of the left ventricular myocardium may fail the myocardial insufficiency in this case being the result of a marked coronary insufficiency. This is evidenced in the electrocardiogram by marked left axis deviation, low voltage of all the leads (QRS, T) or the ST's in Leads 1 and 4 or 2 and 3, instead of high take off or elevation may be markedly depressed, concave, and fused with T's. Clinically there will be dyspnea as the result of the left ventricular failure.

In other cases the myocardial infarct may be the focus for cardiac irritability and cause appearance of premature systoles or paroxysmal auricular flutter as described in the following case.

D. I., male white, aged 58, hypertension for the last few years. Three days before the first electrocardiogram was taken patient had his first coronary attack, with sharp precordial pain, fever, leucocytosis and blood pressure drop from 240/120 to 120/80. Two days later patient had a sudden onset of paroxysmal auricular flutter 2:1 with auricular rate of 302 and ventricular 176. Digitalis one cct unit (0.1 gm.) four times daily, changed the regular ratio (2:1) into irregular (4:3, 2:1) while quinidine sulfate 0.2 Gm. every six hours reestablished the normal sinus rhythm. Exactly a year later a second coronary attack with sharp precordial pain, fever, leucocytosis and marked drop in blood pressure occurred with reappearance of paroxysmal 2:1 auricular flutter. Quinidine reestablished a normal sinus rhythm next day. Patient was well for two years when the third coronary attack occurred with sudden death.

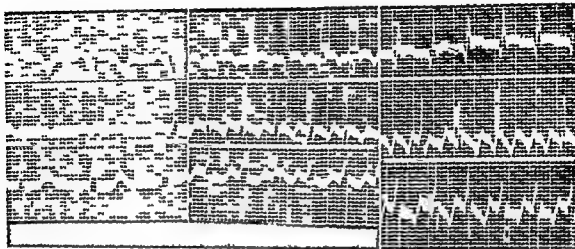
**Summary**—A case of myocardial infarction with paroxysmal auricular flutter is presented.

# PLATE 47 MYOCARDIAL INFARCTION WITH PAROXYSMAL AURICULAR FLUTTER

6/8/33 Aged 53 BP 240/10

6/10/35 BP 120/80

6/13/35 Rate 116 to 176



Q present T inverted coved  
T inverted low

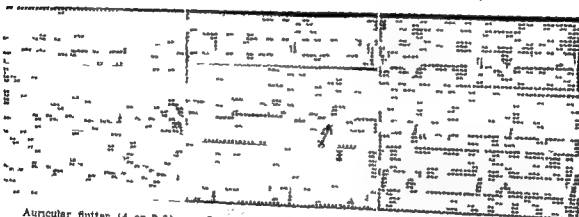
Auricular flutter (2:1) Rate  
116 STs elevated Acute an  
terior infarct

Auricular flutter (2 or - 1)  
Digitalis given

6/1/35

6/20/35

6/3/35



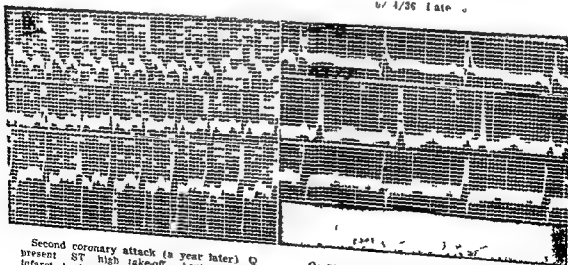
Auricular flutter (4 or 2:1)  
Quinidine given

Regular sinus rhythm Oc  
casional auricular premature  
systole

Similar to 6/8/35

6/23/35 Rate 150

6/4/36 Rate 150



Second coronary attack (a year later) Q  
present ST high take-off Acute anterior  
infarct Auricular flutter (2:1) Quinidine given

Q present STs near base line suggesting  
improved conduction Regular sinus rhythm

## PLATE 48 CORONARY DISEASE WITH A COMPLETE AURICULOVENTRICULAR HEART BLOCK

A W, male white aged 56 Died on Nov 9, 1945

While in good health until July, 1939, patient developed a sudden attack of dyspnea without precordial pain, diagnosed as a coronary attack, and he was hospitalized for ten weeks. He felt well until Nov 9 1945, when while punching the time clock at work he dropped dead. No autopsy was performed.

Electrocardiograms taken at various times between July 1939 and August, 1945 showed a gradual development of auriculoventricular block from a prolonged PR and occasional dropped beat in July 1939 to a complete block in May 1941 with a persistence of a complete block until death in November 1945.

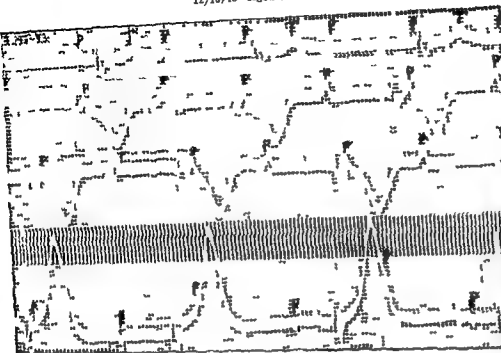
The presence of a complete auriculoventricular block indicated disturbance in conduction in the His bundle and septum involvement in addition to that of ventricular myocardium proper (inverted T's). The striking feature in this case is the unusual high voltage of T's found accidentally once on Dec 13 1943 while patient was feeling fine and the electrocardiograms taken prior to and after this did not show this abnormality.

**Summary**—A case of a coronary disease with a complete heart block is presented. Unusual high voltage of T's was once noted.

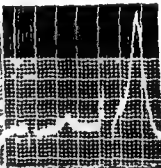
In complete auriculoventricular heart block no impulses reach the ventricles from the auricles. The ventricles respond to a center situated below the block either in the His bundle with QRS and T normal in form and shape or in the ventricles proper with markedly aberrant QRS and T. The aberrant ventricular complexes are similar to those in bundle branch block or ventricular premature systoles. For comparison a single premature systole with high and aberrant T in Lead 4 of another case is shown and similarity with T's of above cited case is noted. Therefore unusually high T's are not an indication of myocardial impairment but due to abnormal initiation and conduction of ventricular impulses in complete auriculoventricular heart block.

# PLATE 48 CORONARY DISEASE WITH A COMPLETE AURICULOVENTRICULAR HEART BLOCK

12/10/43 Aged 54



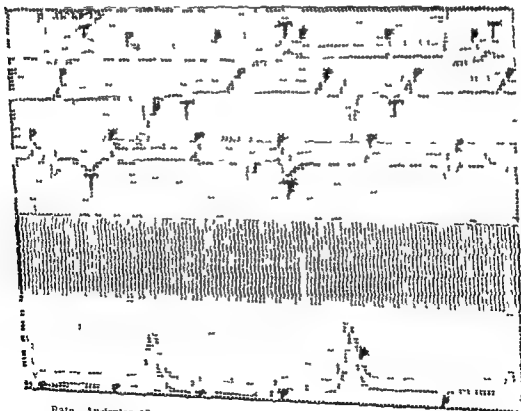
S S female white  
aged 53



Rate Auricular 65 ventricular 88 Complete heart block T<sub>1</sub> up ( mm ) T<sub>2</sub> deeply inverted (12 mm) T<sub>3</sub> up (20 mm) Posterior coronary disease - No complaints Patient ambulant

Lead 4 Single ventricular premature systole with high T similar to T<sub>1</sub> to this ECG

1<sup>st</sup>/9/47 The same patient



Rate Auricular 65 ventricular 0 Complete heart block Note the striking changes which occurred within two weeks - No complaints Patient ambulant

## IV

### HYPERTENSIVE HEART DISEASE

Hypertension is only a symptom and not a disease entity. There are many causes for hypertension, some of which are still unknown. A thorough examination of the patient with complete correlation of history, physical findings, including eye ground examination, and various laboratory procedures, as roentgenograms, electrocardiograms, blood chemistry, urinalyses, and kidney function tests, may help the clinician to arrive at certain diagnostic conclusions, which would be of great importance for the treatment of the patient's hypertension.

In hypertension, i.e., increased tension in peripheral arteries, the peripheral resistance, i.e., diastolic pressure is high. To overcome the high peripheral resistance, the power of the heart, i.e. systolic pressure, must be increased. This will manifest itself also by hypertrophy of the left ventricle, as shown on the roentgenogram and the electrocardiogram with left axis deviation or left ventricular strain. The high systolic pressure and the left ventricular hypertrophy are compensatory mechanisms for high peripheral resistance. The hypertrophy reaches a certain level, beyond which it cannot go and dilatation of the left ventricle sets in, with myocardial insufficiency, dyspnea, and left cardiac failure as physiological and clinical evidence of it. At this stage of cardiac failure and myocardial insufficiency in hypertension we are dealing with hypertensive cardiovascular disease. If unrelieved, the left ventricular failure with dyspnea will be followed by that of the right, with various peripheral edema, ascites, enlarged liver, hydrothorax, and, finally, death.

The kidney plays an important role, and glomerulonephritis is one of the causes of hypertension. In estimating the anatomical and physiological impairment of the kidneys, the physician should not be satisfied with urinalysis and findings of albumin and casts on estimating the albumin globulin ratio and retention of nitrogenous substances in the blood but should perform kidney function tests.

such as Mosenthal's concentration with clearance, dye excretion phenol-sulphonphthalein (PSP) to find out if there is any kidney insufficiency. If there is, and associated with myocardial insufficiency is above we are dealing with cardiovascular renal disease.

Hypertension is frequently associated with arteriosclerosis of peripheral blood vessels including renal. This nephrosclerosis may cause traces of albumin and occasional hyaline casts in the urine but otherwise it is asymptomatic and should not be confused with glomerulonephritis. In renal arteriosclerosis the intima of the blood vessels is affected showing atheromatous plaques and ulcerations but the arterial lumen is patent.

The arterioles of the kidney, as in any other organ of the body, may show a marked concentric thickening of the media the increase in thickness due chiefly to hyalinized fibrous tissue so that in places the lumen is completely obliterated and cannot be identified. This renal obliterative endarteritis is associated with hypertension and may cause renal insufficiency, similar to myocardial insufficiency by coronary occlusion.

If endarteritis affects the pulmonary arteries, or if any pulmonary disease is emphysema, asthma, fibroid tuberculosis cause obstruction in pulmonary circulation we are dealing with pulmonary hypertension in contradistinction to systemic hypertension in systemic blood vessels. To overcome the pulmonary hypertension the right ventricle hypertrophies and establishes a condition known as *cor pulmonale*. The right ventricle may fail and cause cardiac failure and death.

Dilatation of the pulmonary artery and its branches due to increased pressure can be shown on the roentgenogram (see Plate 56) bulging of the left cardiac border in the region of the pulmonary artery and increased hilar shadows due to dilated secondary branches of the pulmonary artery. The right ventricular hypertrophy will cause right axis deviation or if marked right ventricular strain in the electrocardiogram.

The systemic hypertension causes hypertrophy of the left ventricle while pulmonary hypertension that of the right. The condition can be recognized by changes on the roentgenogram while the electrocardiogram shows left or right axis deviation or ventricular strain.

## PLATE 49 HYPERTENSIVE HEART DISEASE WITH LEFT AND RIGHT CARDIAC FAILURE

F. S., male, white, aged 56. Admitted on Nov. 14, 1938, died on Jan. 9, 1939.

Hypertension and severe attacks of dyspnea on exertion of four years' duration. For the last two years, he had increased dyspnea with more orthopnea and he had also noticed swelling of the ankles, he had never had any precordial pain. In the last year he was given mercurial diuretics intravenously as needed, with some improvement of dyspnea and edema.

Examination on admission revealed an obese edematous, dyspneic, orthopneic man who was propped up in bed and sweating profusely, the skin of the face was flushing. There was edema over the sacral and lumbar spines, and slight edema of the scrotum. Few crackling rales over both bases of the lungs. Blood pressure 180/110, pulse 84 regular. The left heart border in the anterior axillary line accentuated, no murmurs. The abdomen was obese and flaccid. The liver was four fingerbreadths below the costal arch, not tender. The diagnosis was hypertension with left cardiac hypertrophy and left and right cardiac failure. On January 9 at 9:30 P. M. he was sitting on the edge of his bed without respiratory difficulty but confused mentally and with slight hallucinations. At midnight the patient was found dead.

**Autopsy**—Marked hypertrophy of the right and left ventricles. Slight acute and chronic passive hyperemia of the lungs, marked passive hyperemia of the liver, kidneys, spleen and gastrointestinal tract. Right hydrothorax, slight ascites, marked increased edema of the brain. Moderate generalized arteriosclerosis.

**Summary**—A case of hypertensive heart disease with left and right cardiac failure is presented. Death due to advanced heart failure.

PLATE 49 HYPERTENSIVE HEART DISEASE WITH LEFT  
AND RIGHT CARDIAC FAILURE



1/ 6 38 Markedly increased cardiac shadow as in cardiac failure Increased lung markings (cardiac)



Marked hypertrophy of the left ventricle The heart with the aorta attached weighed 800 grams



## PLATE 50 HYPERTENSION

Illustrating roentgenographic and electrocardiographic changes in hypertension with left ventricular hypertrophy

**Roentgenogram**—Compare with Plate 1 and note that a normal left ventricular border is straight while in left ventricular hypertrophy the border is rounded. The rounding of the border is due to elongation and hypertrophy of the muscle fibers and is the same in any left ventricular hypertrophy of whatever cause it may be: hypertension, aortic insufficiency or stenosis of rheumatic, arteriosclerotic or syphilitic etiology.

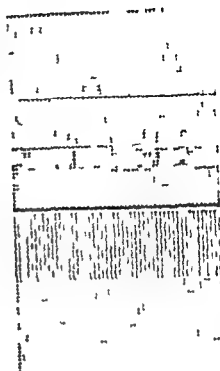
Compare also with Plate 12, anterior view.

**Electrocardiogram**—The left ventricular hypertrophy manifests itself in the electrocardiogram in mild cases by left axis deviation and in marked cases by left ventricular strain. Left axis deviation with high wide slurred QRS, ST<sub>1</sub> depressed and fused with inverted T<sub>1</sub> and often with similar changes in Lead 4. The left ventricular strain is an indication of a mixed hypertrophy and not of a damaged myocardium. Dilatation and failure of the left ventricle with dyspnea may follow this stage of ventricular hypertrophy (see Plate 51).

# PLATE 50 HYPERTENSION

J W male white aged 63

Aged 63 111 - 0/110

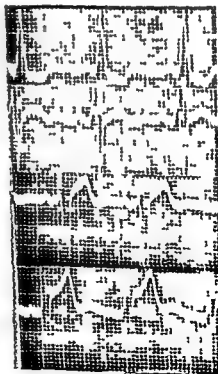


QRS inverted left axis deviation

Left ventricular border is slightly rounded Some calcification of the aortic arch

J A male white aged 68

Aged 68 100/110



Left ventricular strain  
Left axis deviation QRS wide  
high peaked ST<sub>1</sub> depressed and  
fused with inverted T<sub>1</sub>

Left ventricular border is markedly rounded Marked hypertrophy of  
outflow tract left ventricle

## PLATE 50 HYPERTENSION

*Illustration roentgenographic and electrocardiographic changes in hypertension with left ventricular hypertrophy*

**Roentgenogram**—Compare with Plate 1 and note that a normal left ventricular border is straight while in left ventricular hypertrophy the border is rounded. The rounding of the border is due to elongation and hypertrophy of the muscle fibers and is the same in any left ventricular hypertrophy of whatever cause it may be hypertension aortic insufficiency or stenosis of rheumatic arteriosclerotic or syphilitic etiology.

Compare also with Plate 12 anterior view

**Electrocardiogram**—The left ventricular hypertrophy manifests itself in the electrocardiogram in mild cases by left axis deviation and in marked cases by left ventricular strain. Left axis deviation with high wide slurred QRS, ST<sub>1</sub> depressed and fused with inverted T<sub>1</sub> and often with similar changes in Lead 4. The left ventricular strain is an indication of a marked hypertrophy and not of a damaged myocardium. Dilatation and failure of the left ventricle with dyspnea may follow this stage of ventricular hypertrophy (see Plate 51).

PLATE 51 HYPERTENSION WITH LEFT VENTRICULAR  
FAILURE AND ARTERIOSCLEROSIS



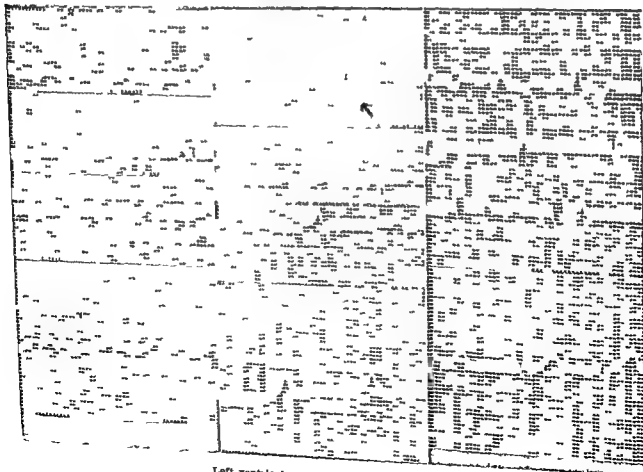
Left ventricular hypertrophy and dilatation Wide aorta  
Left cardiac border is rounded and out

Left oblique view Left ventricular border over  
lapping the spine Diffusely dilated aorta

4/7/48 Aged 60 BP 180/110

3/3/49

1/ 0/40



Left ventricular strain Coronary disease

## PLATE 51 HYPERTENSION WITH LEFT VENTRICULAR FAILURE AND ARTERIOSCLEROSIS

S F, male, white, aged 62

Attacks of nocturnal dyspnea due to left ventricular failure, in the last two and one half years occurring about every three to four months. Blood pressure, 180/110. Pulse, 80 regular except for ventricular premature systoles. Heart markedly enlarged to left with wide aorta. A<sub>2</sub> accentuated no murmurs heard. Urine albumin 2 plus occasional casts. Blood chemistry, normal.

**Roentgenogram, Anterior View**—The left cardiac border is rounded and goes out to the left due to hypertrophy and dilatation of the outflow tract of the left ventricle (anterior portion from apex to aortic valve). The aorta is diffusely enlarged, the upper right cardiac border is bulging convex due to dilated ascending aorta, aortic knob is prominent and there is a lineal shadow descending from the aortic knob to the left of the pulmonary artery due to dilated descending aorta.

**Roentgenogram, Left Oblique View**—As the greater part of the left ventricle is situated posteriorly and cannot be seen on the anterior view to visualize it the heart is rotated clockwise around the longitudinal axis placing the patient in left oblique position. The spine is on the left side and normally the left ventricle inflow tract (posterior portion from mitral valve to apex) just touches the spine. In this roentgenogram the left ventricle is markedly overlapping the spine thus visualizing the marked hypertrophy and dilatation of the left ventricle inflow tract in addition to that of outflow tract as seen in anterior view. Note the diffusely and markedly dilated aorta due to arteriosclerosis.

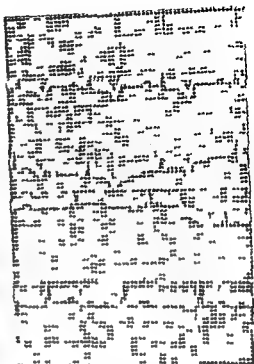
**Summary**—A case of hypertension with left ventricular failure attacks of nocturnal dyspnea and arteriosclerosis is presented to demonstrate characteristic roentgenographic changes in the anterior and left oblique views.

PLATE 52 GLOMERULONEPHRITIS WITH HYPERTENSION  
AND UREMIC PERICARDITIS

3/9/35 H1 240/100



Left ventricular hypertrophy (1 mm thick) Pericarditis slight  
athero clero is of the aortic arch



Left ventricular strain Left axis deviation with high slurred QRS and inverted T1 No history of myocardial infarction



The surface of both kidneys is granular section The cortex has undergone marked distortion and atrophy varying 2 to 5 mm in thickness Some distortion of pelvis  
Histology Chronic glomerulonephritis with a marked decrease in number of glomeruli and a marked degree of sclerosis of the intralobular arteries

## PLATE 52 GLOMERULONEPHRITIS WITH HYPERTENSION AND UREMIC PERICARDITIS

D. D., female, white, aged 43. Admitted on Feb. 20, 1938, died on March 31, 1938.

Headaches and dizziness of two years' duration. Examination made three months before admission showed urine, 1 plus albumin, and occasional red cells. blood urea nitrogen, 57.8 mg. per cent; normal blood count, blood pressure 240/126. Blood pressure normal five years before admission.

On admission, the eye grounds were normal except for vascular tortuosity of the retinal vessels. Heart enlarged to the left, apex beat forcible, rate, 90 regular, blowing systolic murmur heard over the entire precordium but maximal in mitral area. A loud and snappy. Blood pressure 240/120. Electrocardiogram left ventricular strain. Urine specific gravity 1.014, albumin 3 plus, few erythrocytes. Blood hemoglobin 33 per cent, red cells 2,300,000 (anemia), white cells 8,000. Urea nitrogen 87.4, uric acid, 7.9, creatinine 6.2, nonprotein nitrogen 165.5 thus indicating a retention in the blood of renal excretory substances. Carbon dioxide combining power decreased to 40.7 (altered acid base balance). Chlorides 544. Phenolsulfonphthalein (P. S. P.) test returned only 10 per cent in one and one half hours (decreased excretion of dye).

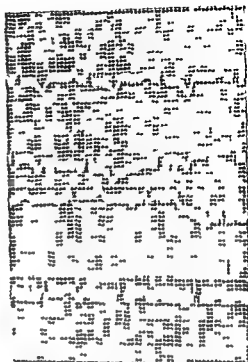
Patient's course in the hospital was characterized by nausea and vomiting, scanty urinary output, high nitrogen in the blood. Three weeks after admission a precordial friction rub was heard for several days, uremic pericarditis. Her condition continued slowly downhill.

**Autopsy**—Chronic glomerulonephritis. Left ventricular hypertrophy (22 mm. in thickness). Anasarca, bilateral hydrothorax, ascites. Chronic passive hyperemia of the liver and spleen. Moderate arteriosclerosis of abdominal aorta, considerably less in the root and arch. Slight coronary sclerosis. Small organizing myocardial infarcts. Slight acute myocarditis, acute serofibrinous hemorrhagic pericarditis. Bronchopneumonia.

**Summary**—A case of glomerulonephritis with hypertension and uremic pericarditis is presented. Death due to renal insufficiency (uremia).

# PLATE 52 GLOMERULONEPHRITIS WITH HYPERTENSION AND UREMIC PERICARDITIS

2/9/28 111 40/1 0



Left ventricular strain Left axis deviation with high slurred Q's and inverted T's No history of myocardial infarction

Left ventricular hypertrophy (1 mm thick) Pericarditis slight



The surface of both kidneys is granular Section The cortex has undergone marked distortion and atrophy varying 3 to 5 mm in thickness Some distortion of pelvis Histology Chronic glomerulonephritis with a marked decrease in number of glomeruli and a marked degree of sclerosis of the intralobular arteries



## PLATE 53 REACTIVATION OF GLOMERULONEPHRITIS WITH HYPERTENSION

D D, male, white, aged 36. Admitted on April 3, 1940, died on April 16, 1940

Pneumonia in November, 1925, followed by nephritis. He felt well until a few months before admission when he began having severe headaches and nose bleed. Two weeks before admission he noticed impaired vision in his left eye. On admission blood pressure 228/128, heart enlarged to left soft blowing apical murmur due to anemia, A<sub>2</sub> equal to P<sub>2</sub>. Red cell count, 2 500 000 hemoglobin, 51 per cent, urine specific gravity 1 008, alkaline albumin + plus 120 red cells, 2 granular casts per cubic millimeter. Urea nitrogen, 53 total nonprotein nitrogen, 70

**Fundi**—Bilateral hypertensive neuroretinopathy with narrowing of retinal arterioles and arteries. Numerous small hemorrhages. The anemic pallor of the discs, opaqueness of retinæ, fluffy white exudates would suggest a nephritic origin of the eye ground lesions rather than a malignant hypertension.

A week after admission, patient complained of substernal discomfort and dyspnea while in bed, red cell count dropped to 2 000 000. Hemoglobin 25 per cent. He died rather suddenly of acute pulmonary edema.

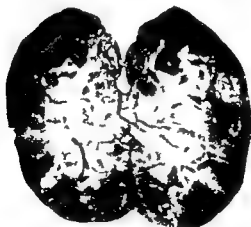
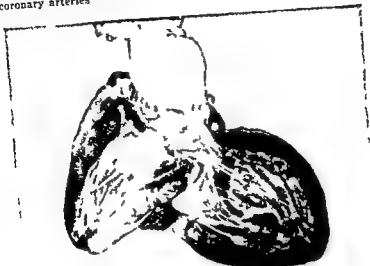
**Autopsy**—Subacute and chronic glomerulonephritis. Hypertrophy of the left ventricle. Moderate atherosclerosis of the aorta. Numerous spontaneous hemorrhages in various parts of the body. Acute hemorrhagic bronchopneumonia.

**Summary**—A case of reactivation of glomerulonephritis is presented accompanied by hypertension and eye ground changes. Numerous spontaneous hemorrhages in various parts of the body with severe anemia. Death due to hemorrhagic bronchopneumonia.

# **PLATE 53 REACTIVATION OF GLOMERULONEPHRITIS WITH HYPERTENSION**

D D male white aged 36 Blood pressure 223/113

The heart with the aorta weighs 609 grams The wall of the left ventricle is hypertrophied measuring 18 mm in thickness at the level of the mitral ring No changes in the valves endocardium myocardium or pericardium Lining of the auricles is smooth and gray Sections of myocardium are tan Moderate atherosclerosis of the aorta and coronary arteries



The initial kidney damage fifteen years ago was not very great since a relatively small number of glomeruli was the seat of a chronic stage

Exacerbation of a chronic glomerulonephritis in the last few months of life caused a subacute involvement of a great number of glomeruli

The degree of renal insufficiency was not very marked

## PLATE 54 HYPERTENSION WITH BENIGN NEPHROSCLEROSIS

W B, male, white, aged 60 Admitted on Dec 20 1940, died on Jan 21, 1941

Dull headaches and tinnitus of two years duration Dyspnea daily for the past two months, with first attack of nocturnal dyspnea a few days before admission Blood pressure, 230/142 The heart enlarged to left, regular rhythm except for occasional premature systole, no murmurs No ankle edema Red blood count, 3,500,000, hemoglobin 70 per cent Urine slightly turbid, specific gravity, 1.012, albumin 4 plus, 30 white and 60 red blood cells per cubic millimeter 2 large granular casts nonprotein nitrogen 56 chlorides, 425 The phenolsulfonphthalein test (P S P) revealed 25 per cent excretion in one and one half hours

Three weeks after admission, patient developed bronchopneumonia Three days before death, nonprotein nitrogen, 83, creatinine, 2.9, carbon dioxide, 77.2, urea nitrogen, 68, chlorides, 440 On the last day, patient became comatose with pulse 132, temperature, 101.8° F abdomen markedly distended by ileus, marked pitting edema of both thighs

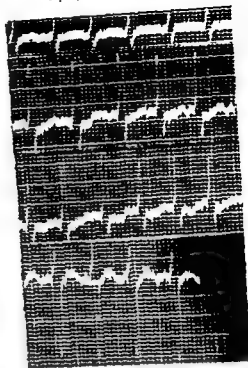
**Autopsy**—Generalized arteriosclerosis Marked benign nephrosclerosis Hypertrophy of the wall of the left ventricle Anasarca of the lower extremities Acute passive hyperemia of liver, stomach spleen and kidneys Acute hemorrhagic ulcerative colitis ileus Bronchopneumonia

Microscopic examination of the kidneys reveals advanced sclerosis of the intralobular and arcuate arteries rather than of the arterioles There are disseminated regions where atrophy of convoluted tubules and groups of glomeruli with acute partial infarction and thrombosis are separated by fairly well intact convoluted tubules and glomeruli

**Summary**—A case of hypertension arteriosclerosis with benign nephrosclerosis is presented Death due to bronchopneumonia

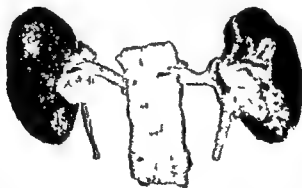
# PLATE 54 HYPERTENSION WITH BENIGN NEPHROSCLEROSIS

1/23/40 R I 20/14



Late 1 1 roxy mal auriculi  
Atrial fibrillation

12/23/40 Wide descending aorta Left ventricular border is rounded  
Increased lung markings from hilum down



Moderate atherosclerosis of the abdominal aorta. Marked benign arteriosclerotic atrophy of the kidneys. Renal arteries although altered by sclerosis have broadly patent lumina. The surface of right kidney is granular. The cut surface of left shows some alteration of the architecture of the cortex.

## PLATE 54 HYPERTENSION WITH BENIGN NEPHROSCLEROSIS

W. B., male, white, aged 60. Admitted on Dec. 20, 1940; died on Jan. 21, 1941.

Dull headaches and tinnitus of two years' duration. Dyspnea daily for the past two months, with first attack of nocturnal dyspnea a few days before admission. Blood pressure, 230/142. The heart enlarged to left, regular rhythm except for occasional premature systole, no murmurs. No ankle edema. Red blood count, 3,500,000, hemoglobin 70 per cent. Urine slightly turbid, specific gravity, 1.012, albumin 4 plus, 30 white and 60 red blood cells per cubic millimeter. 2 large granular casts, nonprotein nitrogen 56, chlorides, 425. The phenolsulfonphthalein test (P. S. P.) revealed 25 per cent excretion in one and one half hours.

Three weeks after admission, patient developed bronchopneumonia. Three days before death, nonprotein nitrogen, 83, creatinine, 2.9, carbon dioxide, 77.2, urea nitrogen, 68, chlorides 440. On the last day patient became comatose with pulse 132, temperature 101.8° F. Abdomen markedly distended by ileus, marked pitting edema of both thighs.

**Autopsy**—Generalized arteriosclerosis. Marked benign nephrosclerosis. Hypertrophy of the wall of the left ventricle. Anasarca of the lower extremities. Acute passive hyperemia of liver, stomach, spleen and kidneys. Acute hemorrhagic ulcerative colitis, ileus. Bronchopneumonia.

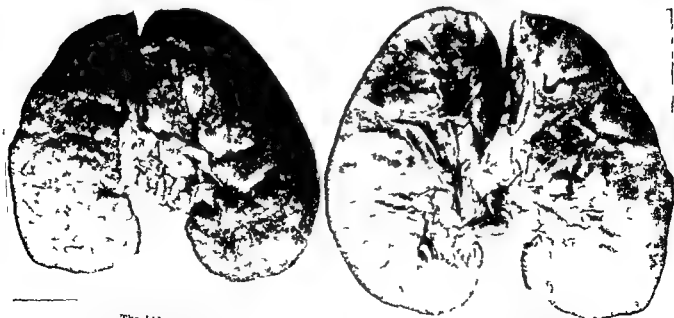
Microscopic examination of the kidneys reveals advanced sclerosis of the intralobular and arcuate arteries rather than of the arterioles. There are disseminated regions where atrophy of convoluted tubules and groups of glomeruli with acute partial infarction and thrombosis are separated by fairly well intact convoluted tubules and glomeruli.

**Summary**—A case of hypertension, arteriosclerosis with benign nephrosclerosis is presented. Death due to bronchopneumonia.

PLATE 55 HYPERTENSION WITH NEPHROSCLEROSIS  
AND ENDARTERITIS



Left ventricular hypertrophy (31 mm thick) Moderate number of small atheromatous plaques in the root of the aorta and aortic valve Coronary arteries unobstructed



The kidney surface is finely nodular The nodules are red purple and yellow tan On cut surface the medulla is dark purple The cortex is to 4 mm thick It is tan with disseminated red and purple points infarcted glomeruli due to occlusion of the small renal arteries

## PLATE 55 HYPERTENSION WITH NEPHROSCLEROSIS AND ENDARTERITIS

I H, male, white, aged 58 Admitted on Nov 4 1939, died on Nov 22 1939

Patient had had hypertension known for three years and albuminuria of unknown duration Failing eyesight in the last two months A month before admission he had an attack of dizziness, perspiration nausea and vomiting lasting for three days Admitted on account of hematuria of one week's duration, nocturia, scanty urination, mild dyspnea constant headaches, markedly impaired vision, tinnitus, nausea, vomiting, restlessness, insomnia, marked weakness Heart enlarged to the left, systolic murmur over precordium Blood pressure, 206/145 no vesicles no edema of the extremities Urine bloody, specific gravity, 1.008; albumin 4 plus numerous red cells few white cells, few granular casts Hemoglobin 80 per cent, red cells 4,150,000, white cells 12,650 Nonprotein nitrogen 56, carbon dioxide 78.2 volumes per cent chlorides, 390

During the next two weeks, patient's condition remained unchanged elevated blood pressure, severe daily headaches, nausea and vomiting, hematuria Five days before death patient had a generalized convulsive seizure, remained semiconscious respirations rapid and shallow, urinary incontinence developed and he became progressively worse

**Autopsy**—Generalized arterio and arteriolar sclerosis Nephrosclerosis Hypertrophy of the wall of the left ventricle 31 millimeters thick heart weighed 625 grams Myocardium firm and tan no areas of infarctions seen except for microscopic scars of the myocardium Acute passive hyperemia of the liver spleen stomach, and kidneys fatty changes of the liver Acute aspiration bronchopneumonia

Microscopic examination of the kidneys revealed a marked atrophy of the convoluted tubules and marked arteriolar sclerosis The walls especially the media of small arteries are tremendously thickened concentrically the increase in thickness due chiefly to hyalinized fibrous tissue occasionally the lumen cannot be identified A similar endarteritis with arteriolar sclerosis present in many other organs

**Summary**—A case of hypertension with nephrosclerosis and endarteritis is presented

# **PLATE 56 COR PULMONALE DUE TO PULMONARY HYPERTENSION AND ENDARTERITIS**

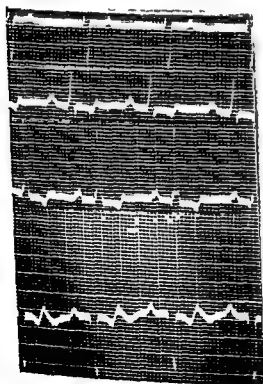


**ARTERIOLAR SCLEROSIS  
ANTEMORTEM FILM**



**Lungs Marked dilatation of the pulmonary artery  
and its branches Extensive arteriolar sclerosis and  
chronic endarteritis**

**S F/41 Aged 40**



**Right ventricular strain Right axis devia  
tion with ST depression T inverted**



**Marked hypertrophy of right ventricle and auricle due to obstruc  
tion in the pulmonary circulation by a chronic pulmonary endarteritis**



## PLATE 56 COR PULMONALE DUE TO PULMONARY HYPERTENSION AND ENDARTERITIS

J. G., male, white, aged 40. Admitted on July 30 1941, died on Aug. 6, 1941

Hemoptysis for two and one half months, cough and blood tinged sputum every morning. Dyspnea on slight exertion. Patient not acutely ill, but cyanotic. Heart and lungs negative. Blood pressure, 120/90. The liver palpable, about four fingerbreadths below the costal margin. Mild clubbing of the fingers. Slight pitting edema of the feet. Temperature normal. Hemoglobin 14 Gm. red cells 4,500,000. white cells, 12,600. Kahn negative.

Two days before death, pulse 80, respirations quiet, muffled venous pulsations in neck, lips cyanotic. Heart apex beat forceful, almost in anterior axillary line blowing systolic apical murmur. Blood pressure 120/80. Tentative diagnosis cor pulmonale probably due to progressive disease of the pulmonary arteries. Two days later patient very restless quite cyanotic, pulse of poor quality respirations shallow and rapid 32 per minute. White count 21,600. The patient grew gradually worse and died that evening.

**Autopsy**—Extensive intimal sclerosis and obliterative endarteritis and moderate atherosclerosis of the pulmonary arteries. dilatation of the pulmonary arteries. embolism thrombosis of the right pulmonary artery. Hypertrophy and dilatation of the walls of the right cardiac muscle and ventricle, enlargement of the cervical veins. Hydrothorax (right 50 cc.) hydropneumothorax (200 cc.) ascites (400 cc.) chronic passive hyperemia of the liver and kidneys. Atherosclerosis of the coronary arteries. fatty changes of theorta. Clubbing of the fingers.

**Summary**—A case of pulmonary endarteritis with hypertrophy and dilatation of the walls of the right cardiac chambers is presented with characteristic changes in roentgenogram and electrocardiogram.

The electrocardiogram of right ventricular strain indicates a marked right ventricular hypertrophy. The precordial electrode placed on the intersection of the left fifth interspace and midclavicular line normally would indicate changes if any in the apical portion of the left ventricle. Here due to marked right ventricular hypertrophy the precordial lead in the above location shows changes in the right ventricle similar to those in Leads 2 and 3. Compare with electrocardiogram and roentgenogram Plate 66.

# PLATE 57 SYPHILITIC HEART DISEASE



Syphilitic aortitis aortic endocarditis and coronary arteritis Obliteration of the mouth of the right coronary artery with small scars in the posterior wall of the left ventricle Moderate hypertrophy of the left ventricle and chronic dilatation of the left ventricle and mitral ring Extensive right hydrothorax chronic passive hyperemia Generalized arteriosclerosis

M. W. male Negro aged 39 Chancre at age 18 treated for six months Wassermann and Kahn positive Dyspnea Orthopnea and swelling of the ankles for ten months Blowing systolic mitral and aortic murmurs Blood pressure 118/84 Right hydrothorax swollen liver edema of the ankles

## V

# SYPHILITIC HEART DISEASE

## PLATE 57 SYPHILITIC HEART DISEASE

Syphilis of the heart and aorta affects the aortic valve, ostia of the coronary arteries, and proximal portion of the aorta

The syphilitic aortitis involves the media of the aorta, causing perivascular infiltration, inflammation, necrosis and later fibrosis, most marked in the proximal portion of the aorta. The fibrosis in the media soon extends to the intima and marked fibrosis and scarring, wrinkling, and pitting of the proximal end of the aorta is striking. Arteriosclerotic aortitis affects the intima forming atheromatous plaques, ulceration, and calcification, most marked in the distal portion of the aorta with greatest intensity in abdominal aorta

The scarring and deformity of the proximal end of the aorta transforms it into a good resonant chamber and adds a musical bell or tambourlike quality to the aortic second sound and as the scarring causes the aortic valve to close under increased pressure the second sound is accentuated. In hypertension the increased blood pressure causes a snappy closure of the aortic valve and accentuation of the second aortic sound

The syphilitic process in the proximal aorta soon spreads toward the aortic valve causing aortic insufficiency and to the mouths of the coronary arteries interfering with normal coronary circulation and causing coronary disease. Clinically syphilitic aortic insufficiency is similar to that of rheumatic or arteriosclerotic etiology. If the hypertrophied left ventricle fails the cardiac failure unrelieved may lead to death

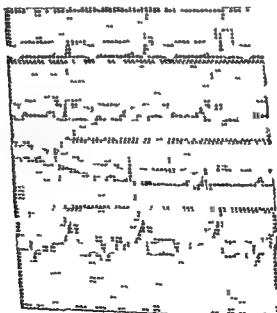
In arteriosclerosis the atheromatous plaques in the wall of the coronary arteries interfere with proper coronary blood supply and thrombosis in the lumen leads to myocardial infarction. In syphilitic coronary arteritis there is no thrombosis but a marked fibrosis and scarring of the mouths of the coronary arteries may lead to coronary occlusion and to myocardial infarction. Syphilis and arteriosclerosis frequently coincide

# PLATE 58 SYPHILITIC AORTIC INSUFFICIENCY, CORONARY DISEASE



Left ventricular border is rounded and out to the left due to left ventricular hypertrophy Note the interlobar fissure of the right lung

1 / 10 / 13 Aged 6



Left axis deviation QRS inverted ST,  
elevated T inverted covered Coronary  
disease

## PLATE 58 SYPHILITIC AORTIC INSUFFICIENCY, CORONARY DISEASE

L. C., male, Negro. Died suddenly at home at 54 years of age.

Chancres at age of 16, syphilis untreated until twenty seven years later. Wassermann and Kahn positive. Development of cardiac symptoms—dyspnea on exertion, occasional nocturnal dyspnea due to left ventricular failure and dull precordial pain at age of 40 at which time the diagnosis of syphilitic aortic regurgitation was made. Double aortic murmur loudest at the third interspace to the left of the sternum apical Austin Flint murmur. Blood pressure 140/44. Corroborative pulse. In addition to signs of aortic regurgitation (murmurs and pulse) patient had pulsating vessels of the neck, chest, arms and capillary pulse as evidence of associated vasodilatation.

The electrocardiogram in 1932 was normal except for left axis deviation. Electrocardiograms taken at various intervals from 1939 to 1943 indicate coronary disease with inverted T's. The question of coronary disease being syphilitic with involvement of the mouths of coronaries or atherosclerotic as expected at patient's age is of academic rather than practical value.

**Summary**—A case of syphilitic aortic insufficiency with coronary disease as shown on the electrocardiogram is presented.

# PLATE 50 SYPHILITIC AORTIC ANEURYSM



Case 1 Ruptured aortic aneurysm



Case 2 Ruptured saccular aneurysm



View from bronchial side.



View from aortic side

Case 3 Saccular aneurysm of aortic arch with erosion into bronchus and fistula

## PLATE 59 SYPHILITIC AORTIC ANEURYSM

A syphilitic aortitis may cause a localized dilatation and bulging, with a sacular aneurysm in the thoracic aorta. Abnormal pulsation may be noted on inspection and a heaving pulsation on palpation over the upper anterior chest, with expansile quality, if the chest has been eroded. Occasionally, an aneurysm fills with fibrin or thrombus and does not pulsate.

Symptoms when present are due to pressure on or erosion of adjacent structures, as blood vessels (inequality of pulse and blood pressure in the two arms) nerves (inequality of pupils, hoarseness) trachea (tracheal tug) bronchus (cough), esophagus (dysphagia), spine (erosion), and ribs (expansile pulsation).

If the aneurysm compresses the innominate artery, the right radial pulse will be weaker than the left, while if the aneurysm lies between the innominate artery and the left subclavian the left radial pulse is delayed and weaker than the right. Similarly, there may be differences in blood pressure in the two arms.

Inequality of the pupils results from pressure on the sympathetics stimulation causing dilatation and paralysis producing contraction. Hoarseness is due to pressure on the left recurrent laryngeal nerve and paralysis of the vocal cords.

When the aneurysmal sac of the transverse arch of the aorta has become adherent to the trachea a tracheal tug may be elicited on palpation of the trachea. Pressure on the trachea may cause diminished breath sounds over lungs. In pressure on a main bronchus the breath sounds are diminished or absent over the affected lung.

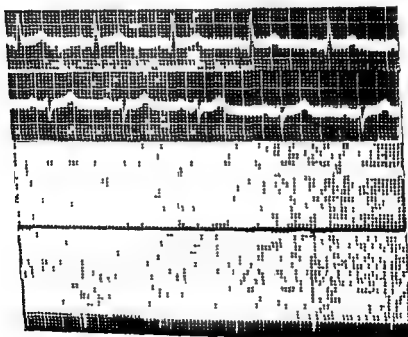
Aortic aneurysm may rupture and cause the patient's death.

# PLATE 60 SYPHILITIC AORTIC ANEURYSM



10/14/40 Syphilitic aneurysm of the aortic arch with pressure on left bronchus Trachea is displaced to the right

1/5/43 Aged 4: Rate "6



Left axis deviation



## PLATE 60 SYPHILITIC AORTIC ANEURYSM

A V, white, male Died at 47 years of age

Chancre at 21 years of age with a short course of antisyphilitic treatment, but none after that Wassermann and Kahn positive Slight cough with expectoration of seven years' duration, dyspnea on exertion two years, no hoarseness An abnormal chest condition was found on pre employment physical examination

Pulse, 76, regular, equal in both wrists Blood pressure, 130/70 (left), 124/70 (right) Bulging and slight pulsation of the left upper chest, right chest is flat, prominent heaving apex impulse noticed to the left of the mid clavicular line, also noticeable suprasternal pulsation and bulging No murmurs Tracheal tug present in the neck Due to pressure of the aneurysm on the left bronchus, the left upper lung dull anteriorly and posteriorly, breath sounds are distant, nearly absent no tubular breathing, no amphoric sounds no râles cough and dyspnea

Patient died suddenly (rupture of the aneurysm?) at home on Oct 28, 1943 No autopsy

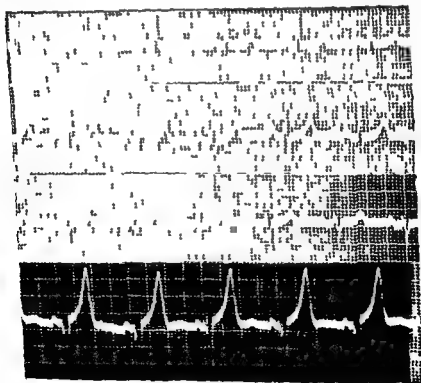
**Summary**—A case of syphilitic aortic aneurysm with pressure on the left bronchus is presented

PLATE 61 SYPHILITIC AORTIC ANEURYSM



1/16/43 Syphilitic aneurysm of the aortic arch with pressure and erosion of thoracic vertebrae

1/18/43 Aged 59



Left axis deviation

## PLATE 61 SYPHILITIC AORTIC ANEURYSM

G P, white, male Died at 58 years of age

Chancro at 29 years of age, no treatment Twenty years later, dyspnea on slightest exertion Wassermann and Kahn positive Blood pressure, 140/60 Examination revealed syphilitic aneurysm of the aortic arch with aortitis, aortic insufficiency, and early cardiac failure Three years later (in 1940) dyspnea on exertion and severe pain in the back in the region of the thoracic spine Roentgenogram of the thoracic spine in 1943 revealed an erosion of the anterior and left borders of the fourth fifth and sixth thoracic vertebrae, secondary to a large aortic aneurysm On April 4 1944 we have following notation in our records "Patient coughs constantly, very short of breath marked weakness, the pain in front of the chest and the back is worse and extends downward into upper abdomen Heart to and fro murmurs at the base Lungs moist and dry rales Tracheal tug Roentgenogram of the thoracic spine reveals further involvement and the bodies of two vertebrae are two thirds destroyed It is difficult to predict outcome but patient may have a sudden collapse at any time either from collapse of fourth fifth or sixth vertebrae and compression of cord (life is seldom possible when this occurs at this level) or rupture of the aneurysm" Two weeks later patient died suddenly at home No autopsy

**Summary**—A case of syphilitic aortic aneurysm with pressure and erosion of thoracic vertebrae is presented

practically reversing the normal relationship of the left and right heart as to the work performed

Defect in the interventricular septum is usually in the membranous, weakest part of the septum. The arterial blood from the left ventricle will flow into the right and mix with venous blood. There will be no cyanosis, and a systolic murmur in the third or fourth interspace to the left of the sternum may be the only evidence of septum defect. The same is true of acquired ventricular septum defect (Plate 40)

Pulmonary stenosis, if it occurs alone, without a ventricular septum defect, causes a systolic murmur in the pulmonary area, right ventricular hypertrophy, but no cyanosis, as there is no defect in the septum and no mixture of arterial and venous bloods will occur

In the tetralogy of Fallot, in addition to pulmonary valve, or infundibular stenosis, there is interventricular septum defect, right ventricular hypertrophy, and dextroposition of the aorta. The tetralogy of Fallot is an example of venoarterial shunt, venous blood of the right ventricle mixing with arterial blood of the left causing a moderate to marked cyanosis. This kind of mixture is possible because of dextroposition of the aorta and ventricular septum defect with markedly increased pressure in the right ventricle over that of the left caused by pulmonary stenosis and hypertrophy of the right ventricle

The venoarterial shunt cyanosis should not be confused with cyanosis of right ventricular failure, peripheral stasis, or that of pulmonary origin, but in any cyanosis of whatever origin the abnormal color of the blood made visible by dilated capillary vessels is due to the increased amount of reduced hemoglobin

In venoarterial shunt a large number of red cells do not come in contact with oxygen in the alveoli of the lungs, and therefore there is insufficient oxygenation. As compensatory physiological adjustment, the oxygen capacity is increased by increasing the number of red cells, polycythemia, thus supplying sufficient oxygen to the tissues, with a result that the amount of oxygen in the blood is nearly normal

## CONGENITAL ANOMALIES OF THE HEART AND LARGE BLOOD VESSELS

Congenital anomalies are various defects in the structures of the heart and large blood vessels. When an anomaly causes a cardiac failure, we may call it then a congenital heart disease. Only a few of these anomalies which are compatible with life, and of clinical importance, are to be considered here.

It is customary to include congenital dextrocardia in the discussion of congenital anomalies, although it is a normal heart, but congenitally transposed, with defect in the position and not in the structure of the heart.

Congenital dextrocardia and coarctation of the aorta are examples of congenital anomalies without abnormal communication or shunt between the right and left side of the heart.

Patent ductus arteriosus, atricular and ventricular septum defects, are examples of arteriovenous shunt with arterial blood entering the venous circulation.

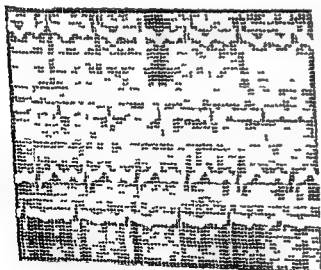
The atricular septum defects are (1) the patent foramen ovale, a frequently found anomaly and of no clinical importance (Plate 63), (2) persistent ostium primum (Plate 67), embryologically, the original opening in this part of the septum closes first, and (3) persistent ostium secundum, the original opening in the part of the septum in which foramen ovale is located, closes second, called also widely patent foramen ovale (Plate 66).

As the left auricle is situated anatomically above the right, the arterial blood of the left auricle in widely patent foramen ovale (Plate 66) enters the right auricle and mixes with its venous blood. To take care of the extra load the right ventricle with tricuspid and pulmonic valves, the pulmonary artery and its branches will markedly hypertrophy. The left ventricle and aorta will be small, hypoplastic, thus

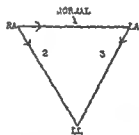
# PLATE 62 CONGENITAL DEXTROCARDIA (TRANPOSED HEART)



Cardiac apex and aortic knob on the right side. Lipiodol visualizes transposition of the straight right and slant left bronchus

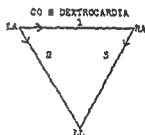


Lead 1 (P QRS T) invert d  
Leads 2 and 3 replace each other  
Lead 4 inverted and similar to Lead 1



Lead - Normal

- 1 Right to left forearm  
RA → LA
- Right arm to left leg  
RA → LL
- 3 Left arm to left leg  
LA → LL



Leads -

## Congenital dextrocardia

- 1 LA → RA  
Lead 1 reversed
- 2 LA → LL  
Actually Lead 3 is taken
- 3 RA → LL  
Actually, Lead 2 is taken

## PLATE 62 CONGENITAL DEXTROCARDIA (TRANSPPOSED HEART)

M M, female, white, aged 29, no cardiac complaints

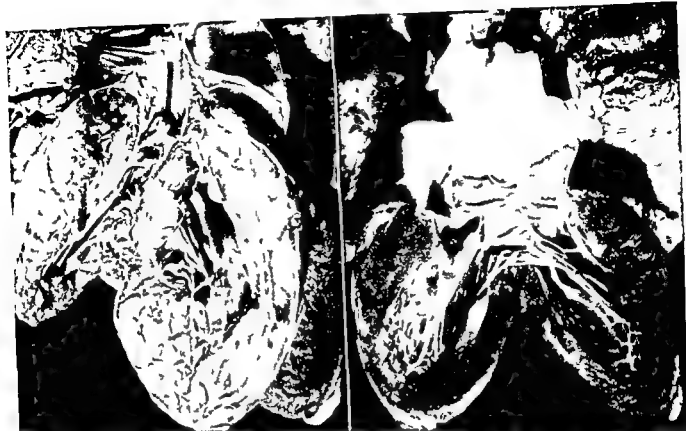
**Roentgenogram**—Cardiac apex and aortic knob on the right side, as the heart is a mirror picture of a normally placed heart. The gas in the stomach may be seen on the right side, as transposed heart is usually associated with transposition of abdominal organs. The situs inversus involves also the bronchi and the lungs. Lipiodol visualizes transposition of the straight right and the slant left bronchus. See Plate 59, photograph of a trachea, straight right and slant left bronchus.

**Electrocardiogram** shows characteristic changes due to transposition. Leads 1 and 4 completely inverted, Leads 2 and 3 replace each other. The electrocardiogram of the first three leads is similar to that which is found when lead wires of both arms are crossed, but Lead 4 in later instance is normal, not inverted.

In acquired dextrocardia (see Plate 5) i.e., right sided, displaced, and not transposed heart—as found in pneumothorax, fluid, or adhesions—the apex, aortic knob, and gas in the stomach are on the left side. The electrocardiogram is normal.

Dextroversio cordis (Plate 62) is an arrested development in early embryonic life when the heart did not complete its version or rotation from right to left, and remained on the right side. The electrocardiogram is normal. As dextroversio cordis is a developmental defect it is often associated with other congenital cardiac defects.

PLATE 63 DEXTROVERSIO CORDIS, PULMONARY STENOSIS,  
SEPTUM DEFECTS



Marked stenosis of pulmonary valve Hypoplastic  
pulmonary artery Interventricular septal defect Right  
ventricular hypertrophy

Hypertrophy and dilatation of the aorta Closed  
ductus arteriosus Interventricular septal defect



Patent foramen ovale



## PLATE 63 DEXTROVERSIO CORDIS, PULMONARY STENOSIS, SEPTUM DEFECTS

R. M., male, white, aged 5½. Died on admission on March 8, 1943

At one month of age, patient had cyanotic lips and diagnosis of dextro cordis was made. The child had never walked, had started to talk at age 4, and was mentally retarded. He was apparently well until one week before admission, when he developed measles, and then bronchopneumonia three to four days later. Physical examination on admission revealed irregular, slow respirations, measles, cyanotic lips, feeble heart tones. Patient died five minutes after admission.

**Autopsy**—Marked stenosis of the pulmonary valve, with hypoplastic pulmonary artery (poststenotic hypoplasia), and hypertrophy of right ventricle and right auricle. Patent foramen ovale, interventricular septal defect, hypertrophy and dilatation of the aorta with closed ductus arteriosus. Dextroversio cordis with the left side of the heart made of the left ventricle, the apex is that of the left ventricle and points to the right side. Maculopapular rash of the head and trunk (history of recent measles). Hyperemia, edema, and disseminated hemorrhages of the lungs. Acute mucopurulent bronchitis, acute and passive chronic hyperemia of the lungs, liver, abdominal viscera, and brain. Polycythemia and slight dehydration.

**Summary**—A case of dextroversio cordis with pulmonary stenosis, interventricular septal defect and patent foramen ovale in a mentally retarded boy is presented. Death due to bronchopneumonia.

In dextroversio cordis the heart remains on the right side, due to failure of normal version of the heart from right to left. The electrocardiogram is normal and does not show characteristic changes as found in congenital dextro cordis (transposed heart).

# PLATE 64 COARCTATION OF THE AORTA



F H male aged 4 Died of bronchopneumonia

Constriction of the aorta at the site distal to the origin of the left subclavian artery and close to the insertion of the ligamentum arteriosum. The constriction (coarctation) is sharp and annular—as constricted with a cord from outside while in stenosis the lumen is obstructed from within. Note the dilated intercostal arteries arising from the thoracic aorta.

## PLATE 64 COARCTATION OF THE AORTA

The constriction of the aorta is distal to the origin of the left subclavian artery. The blood pressure is increased in the precontracted part of the aorta and arteries of both arms and decreased in postcontracted part of the aorta and arteries of the legs.

A loud systolic murmur heard over the upper sternum and in the left inter-scapular region where the aortic arch is close to the spine.

Collateral circulation is established through various arteries especially the intercostals which become prominent with visible pulsation. The dilated intercostal arteries may cause erosion of the lower borders of the ribs as shown on the roentgenogram.

# PLATE 65 PATENT DUCTUS ARTERIOSUS



E. L. female white aged 36 Died of intra-abdominal hemorrhage following cesarean section The duct is between the left pulmonary artery (at a site close to the bifurcation of the main pulmonary artery) and aorta (at a site distal to the origin of the left subclavian artery) Note the glass rod in the duct The aorta is wide open and cut The pulmonary artery (sewed in here) is markedly dilated

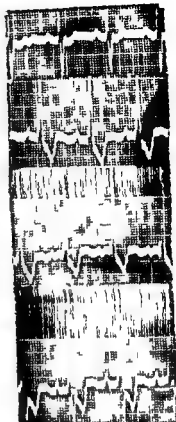
## **PLATE 65 PATENT DUCTUS ARTERIOSUS**

Patent ductus arteriosus is similar to arteriovenous aneurysm (see Plate 33). There is an abnormal communication between the arterial blood vessel the aorta and the venous the pulmonary artery. The blood flows continuously as in arteriovenous aneurysm from the aorta into the pulmonary artery and causes a harsh loud continuous murmur, best heard in the second or third left interspace, or upper sternum and the left interscapular area where the aortic arch is close to the spine. Due to increased blood volume the pulmonary artery is dilated and prominent on the roentgenogram.

# PLATE 66 WIDELY PATENT FORAMEN OVALE AURICULAR SEPTUM DEFECT



Prominent pulmonary artery and cobus. The left lower cardiac border is out rounded and globular due to hypertrophy of the right ventricle. Increased hilar shadows due to dilated secondary branches of the pulmonary artery.



Right ventricular strain. Right axis deviation with high wide QRS in all leads.  $QT_1$  ;  $\epsilon$  markedly depressed, deformed (slanting) and faced with deeply inverted T ;  $\epsilon$



Widely patent foramen ovale. Chronic indurative mitral endocarditis. Hypoplasia of left ventricle and aorta. Note the marked hypertrophy of the right ventricle and the small size of the left.



Marked hypertrophy of the right ventricle. Hypertrophy of tricuspid and pulmonary valves. Hypertrophy and dilatation of pulmonary artery and its branches.

## PLATE 66 WIDELY PATENT FORAMEN OVALE AURICULAR SEPTUM DEFECT

A B, female, white, aged 38. Admitted on Feb 8 1942, died on Feb 19 1942

Pneumonia at 6 years of age, ill for about one month. At 14 years of age, she was told she had leakage of the heart, which was not of recent origin or associated with infection at that time. She was well until the second pregnancy at 25 years of age, when she first noticed dyspnea on exertion. She had a cholecystectomy four years before admission. At that time her heart revealed rough to and fro murmurs at apex and base. A year later, dyspnea on exertion set in with palpitation at times but no edema. On admission, the pulmonary sounds sharply accentuated and greater than aortic sounds. A loud diastolic murmur heard over a large area of the upper sternum. Also tricuspid systolic and a loud mitral systolic murmur transmitted toward the axilla. Blood pressure, 126/90.

Exploratory thoracotomy for a suspected patent ductus arteriosus and possibly its ligation did not reveal any patent ductus. Patient's temperature rose to 103° F. and she died thirty six hours after the operation.

**Autopsy**—Congenitally widely patent foramen ovale marked hypertrophy of the right ventricle 11 millimeters thick with marked hypertrophy of tricuspid and pulmonary valves hypertrophy and dilatation of the walls of the pulmonary artery and its branches slight atherosclerosis of pulmonary artery. Hypoplasia of left ventricle and aorta. Chronic indurative mitral endocarditis. Recent surgical defect of thoracic wall and incision of the pericardium acute serofibrinous pericarditis and left pleuritis compression atelectasis of left lung acute distention of right lung acute dilatation of right auricle. Coronary arteries normal.

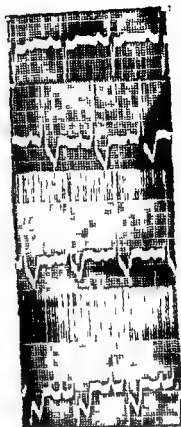
**Summary**—A case of widely patent foramen ovale with hypertrophy of the right ventricle and the pulmonary artery and its branches is presented. Note the characteristic roentgenographic and electrocardiographic changes and compare with Plate 56 of cor pulmonale with pulmonary hypertension.

The precordial electrode placed on the intersection of the left fifth inter space and midclavicular line normally would indicate changes if any in the apical portion of the left ventricle. Here due to marked right ventricular hypertrophy the precordial lead in the above location shows changes in the right ventricle similar to those in Leads 2 and 3.

# PLATE 66 WIDELY PATENT FORAMEN OVALE AURICULAR SEPTUM DEFECT



Prominent pulmonary artery and conus. The left lower cardiac border is out rounded and globular due to hypertrophy of the right ventricle. In the right lower cardiac border shadows due to dilated secondary branches of the pulmonary artery.



Right ventricular strain, right axis deviation with high wide QRS in all leads, ST depression, markedly depressed ST formed (slanting) and deeply inverted T waves.



Widely patent foramen ovale. Chronic inflammatory tricuspid endocarditis. Hypoplasia of left ventricle and aorta. Note the marked hypertrophy of the right ventricle and the small size of the left.



Marked hypertrophy of the right ventricle. Hypertrophy of tricuspid and pulmonary valves. Hypertrophy and dilatation of pulmonary artery and its branches.



## PLATE 67 AURICULAR SEPTUM DEFECT, PERSISTENT OSTIUM PRIMUM

T B, female, white, aged 38 Admitted on Feb 14, 1942, died on Feb 15, 1942

Born prematurely (7 months) 2½ pounds at birth Normal child until 12 years of age, when she began to develop scoliosis of the spine She was always small In good health until eight months before admission, when she began to have dyspnea on slightest exertion and had to curtail her activities On admission pulse, 120 temperature, 99° F respirations 30 weight, 87 pounds blood pressure, 130/100 Small kyphoscoliotic white female Orthopnea and marked cyanosis Heart systolic blowing murmurs at the base and apex Fluoroscopy marked scoliosis with convexity to the right and rotation of the heart counterclockwise as in right oblique position, apex beat displaced to left pulmonary conus is prominent Clinical diagnosis marked kyphoscoliosis with cardiac failure due to torsion of the heart auricular septal defect with cyanosis tardive Patient expired day after admission

**Autopsy**—Congenital auricular septal defect (persistent ostium primum) with deformity of the mitral and tricuspid valves, induration of the mitral tricuspid, and aortic valves marked hypertrophy of the wall of the right ventricle dilatation of the right auricle Acute verrucous mitral and tricuspid endocarditis hydropericardium Hypertrophy and dilatation of pulmonary artery atherosclerosis of pulmonary artery Polycythemia Marked kyphoscoliosis of the spine Chronic passive hyperemia of the lungs liver and kidneys Compression atelectasis of lower lobe of left lung acute distention of right lung bronchopneumonia

**Summary**—A case of advanced heart failure due to marked kyphoscoliosis of thoracic spine is presented associated with congenital auricular septal defect and chronic induration of mitral tricuspid and aortic valves

PLATE 67 AURICULAR SEPTUM DEFECT PERSISTENT  
OSTIUM PRIMUM



Induration of mitral and tricuspid valves



Marked hypertrophy of the right ventricle Indurated  
tricuspid normal pulmonary valve



Induration of the aortic valve

## PLATE 68 INFUNDIBULAR PULMONARY STENOSIS WITH INTERVENTRICULAR SEPTUM DEFECT

E O, male, white aged 22 Systolic thrill and loud systolic murmur at third and fourth interspaces to the left of the sternum Cyanosis dyspnea clubbing of the fingers and toes Polycythemia (red cell count, 6 070 000) with high hemoglobin (118 per cent) Patient died of pulmonary hemorrhage due to a far advanced pulmonary tuberculosis

**Autopsy**—The heart weighs 325 grams The right ventricle (the wall and the trabeculae) is much thickened up to 16 millimeters especially in its posterior and right lateral portions The main portion of the right ventricle communicates with the pulmonary conus (a conuslike part of the right ventricle situated below the pulmonary valve) through a 4 by 2 millimeter oval stenotic orifice The wall of the poststenotic conus is only 2 millimeters thick and does not participate in general hypertrophy of the right ventricle The pulmonary valve and aorta are normal The right ventricle is also in communication with the left ventricle through a patency in the membranous portion of the interventricular septum The left ventricle is of normal thickness 11 millimeters The mitral and aortic valves are normal The aorta is thin walled and somewhat narrowed throughout No dextroposition of the aorta as seen usually in Fallot tetralogy Ductus arteriosus is not patent

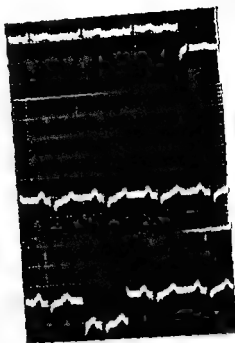
**Summary**—A case of infundibular pulmonary stenosis with interventricular septum defect is presented The venoarterial shunt with cyanosis was due to two factors (1) the septum defect and (2) increased pressure of the right ventricle due to right ventricular hypertrophy so that the venous blood of the right ventricle passed through the septum defect and mixed with the arterial blood in the left ventricle

The stenosis is not in the valve itself but on the tip of the infundibular portion of the right ventricle (pulmonary conus) The systolic murmur in the infundibular pulmonary stenosis is heard best in the third or fourth interspace to the left of the sternum close to the location of the pulmonary conus

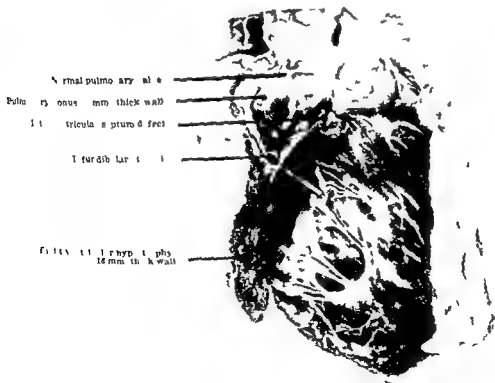
# PLATE 68 INFUNDIBULAR PULMONARY STENOSIS WITH INTERVENTRICULAR SEPTUM DEFECT



Normal cardiac contour Pulmonary tuberculosis left lung



Right axis deviation due to right ventricular hypertrophy



Infundibular stenosis is on the tip of the pulmonary conus and not on the pulmonary valve Interventricular septum defect is in the membranous portion of the septum

## PLATE 69 TETRALOGY OF FALLOT

I E, female, white aged 26 housemaid

Cyanosis since birth with dyspnea on exertion only. Marked cyanosis of the skin, especially hands and feet, also ears, nose, and lips. Marked clubbing of the fingers and toes. Vasodilatation and trophic changes with ulcers in the skin of both lower legs. Heart: a loud systolic murmur heard over entire precordium with greatest intensity in the third and fourth interspace near sternum, suggesting infundibular pulmonary stenosis. Slight scoliosis of the thoracic spine with convexity to the left. Erythrocytes 8,670,000, hemoglobin 163.8 per cent, 27.3 Gm, color index, 1.0, leucocytes, 7,050.

**Roentgenogram**—Heart is slightly globular in shape, probably due to hypertrophy of the right ventricle to the left and right. The aortic knob is visible as increased round density on the right side indenting the barium-filled esophagus to the left, while a normally placed aorta would indent it to the right. The ascending aorta is on the left. The roentgenographic evidence is a completely transposed aorta, while the dextroposition of the aorta in tetralogy of Fallot is usually a slight degree of transposition. The pulmonary conus in infundibular pulmonary stenosis is the poststenotic portion of the right ventricle and is not visible on the roentgenogram because it is atrophic and hypoplastic.

**Discussion**—Tetralogy of Fallot is a congenital anomaly with dextroposition of the aorta, ventricular septum defect, pulmonary (infundibular) stenosis, and right ventricular hypertrophy. The marked cyanosis since birth indicates venoarterial shunt. Red cell count is 8,670,000, hemoglobin, 163.8 per cent, 27.3 grams. The shunt is about 50 per cent. About one half of the venous blood of the right ventricle passes through the pulmonary artery into the pulmonary circulation. The other half enters the aorta directly, due to dextroposition of the aorta and its origin from the right ventricle, and also passes through the ventricular septum into the left ventricle, due to increased right ventricular pressure (hypertrophy).

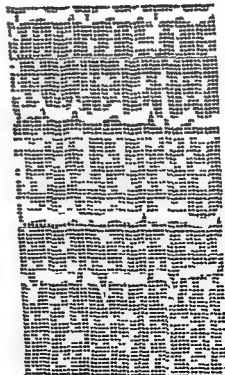
# PLATE 69 TETRALOGY OF FALLOT



Marked cyanosis Clubbing of the fingers



Dextroposition of the aorta Indentation on a barium filled esophagus with convexity to the left



Right axis deviation i. e. high sharply peaked auricular hypertrophy T<sub>1</sub> normally inverted in children and occasionally in young adults

## I NORMAL HEART

The greater portion of the left ventricle and both auricles are located posteriorly, and visualized on the roentgenogram in the left and right oblique views.

In a normally built adult the heart is in an oblique position, while in a tall, thin person it is vertical, and in a stout, short individual it is horizontal. In young children the heart is globular and centrally located.

The electrocardiogram shows variations which are normal for that particular type of heart. In the horizontal heart the electrocardiographic changes are in Lead 3: QRS: low, notched, splintered, diphasic or inverted with T: frequently inverted. In the vertical heart the electrocardiographic changes are in Lead 1: QRS: low, diphasic, or inverted. In a globular heart a right axis deviation is found, which is normal in children.

A normal heart is rotated by scoliosis of the spine. In scoliosis with convexity of the spine to the right, the heart is rotated counter-clockwise, and in the anterior view the heart contour is similar to that of the right oblique view. In scoliosis with convexity of the spine to the left, the heart is rotated clockwise, and in the anterior view the heart contour is similar to that of the left oblique view.

A normal heart may be displaced to the right or left, as by pneumothorax, fluid, or adhesions. Except for a shifting axis, the electrocardiogram is normal in a rotated or displaced heart.

## II RHEUMATIC HEART VALVULITIS, MYOCARDITIS, PERICARDITIS, BACTERIAL ENDOCARDITIS

### 1 Mitral Stenosis

Mitral stenosis causes hypertrophy and dilatation of the left auricle and hypertrophy of the right ventricle. The enlarged left auricle compresses the esophagus and left bronchus and while enlarging anteriorly and to the right pushes and rotates the heart counterclock-

wise on its vertical axis, as shown on roentgenograms in anterior, left and right oblique views. The more marked is the stenosis, the more pronounced is the left auricular enlargement and rotation of the heart on the roentgenogram.

The electrocardiogram may show right axis deviation due to right ventricular hypertrophy and high, peaked P<sub>w</sub> due to auricular hypertrophy. Auricularitis in mitral stenosis may cause abnormal auricular myocardial conduction with deformed P<sub>w</sub> wide, notched high or low, diphasic, or inverted. The anatomical changes in the left auricle may cause increased irritability and formation of auricular premature systole, paroxysmal auricular tachycardia (which is a succession of numerous auricular premature systoles), or, as frequently is the case, auricular fibrillation with abnormal initiation and conduction of auricular impulses.

## 2 Aortic Insufficiency

Aortic insufficiency causes hypertrophy of the left ventricle as shown on the roentgenogram in the anterior and left oblique views. The more marked is the insufficiency, the more pronounced is the left ventricular hypertrophy.

In mild cases, the left ventricular border, in the anterior view, from the end of the pulmonary contour down is rounded and extends below the diaphragm. In left oblique view the left ventricular border is rounded and heart is globular in shape. The electrocardiogram may show left axis deviation and high voltage of QRS.

In marked cases the left ventricular border is not only more rounded but extends out to the left in the anterior view and overlaps the spine in the left oblique view. At this stage a marked hypertrophy of the left ventricle may be followed by dilatation, with the left ventricular border markedly out to the left and overlapping the spine. The electrocardiogram shows left ventricular strain, left axis deviation with high wide slurred QRS, ST<sub>w</sub> depressed and fused with inverted T<sub>w</sub> and often inverted T<sub>r</sub>. The left ventricular strain is an indication of a marked hypertrophy and not of a damaged myocardium. Dilatation and failure of the left ventricle with dyspnea may follow with signs of myocardial insufficiency in the electrocardiogram as tachycardia, low voltage of all leads.

If aortic insufficiency is associated with mitral stenosis the enlarged left auricle may be seen in the right oblique view on the roentgenogram with compression of the esophagus in addition to the left ventricular hypertrophy of aortic insufficiency.



### 3 Pulmonary Stenosis (See Plate 18)

Pulmonary stenosis usually accompanied by mitral stenosis causes a marked hypertrophy of the right ventricle, especially the pulmonary conus i.e. the infundibulum portion of the right ventricle situated below the pulmonary valve. The hypertrophy of the right ventricle and pulmonary conus is more markedly pronounced than in the case of mitral stenosis alone. The roentgenogram shows an enlarged left auricle on the right cardiac border and markedly counterclockwise rotated heart due to mitral stenosis. In addition, a marked bulging and prominence of the pulmonary conus is seen, due to a marked infundibular hypertrophy and pulmonary stenosis, which cannot possibly be caused by mitral stenosis alone.

The electrocardiogram will show right axis deviation and possibly auricular fibrillation as seen in marked mitral stenosis cases, but in addition right ventricular strain is present. Right axis deviation with ST<sub>2</sub>, 3 depressed, T<sub>2</sub>, 3 inverted, indicating a marked right ventricular hypertrophy.

In aortic stenosis or marked aortic insufficiency there is a marked left ventricular hypertrophy as shown on the roentgenogram by marked changes on the left ventricular border and in the electrocardiogram by left ventricular strain. In pulmonary stenosis there is a marked right ventricular hypertrophy as shown on the roentgenogram by marked bulging and prominence of the pulmonary conus of the right ventricle on the pulmonary mid left cardiac contour and in the electrocardiogram by right ventricular strain.

### 4 Myocarditis

In myocarditis without impaired myocardial function the cardiac contour on the roentgenogram is normal. Myocardial insufficiency and cardiac failure is associated with dilatation of cardiac chambers and the heart contour will be enlarged in all directions, affecting all the chambers of the heart. This dilatation may be confused with pericarditis and at times differentiation may be difficult especially when myocarditis is associated with pericarditis. Correlation with history, physical findings, and the electrocardiogram is of great value in such cases.

Myocarditis may cause impaired conduction in the myocardium proper or the auriculoventricular conduction system or both. In contradistinction to myocardial infarction where the myocardial damage is usually localized, the rheumatic myocarditis is generalized of a disseminated type, causing changes in all the leads with disturbances in intraventricular conduction, as shown by wide, slurred, or notched splintered QRS, deformed ST, or low, inverted T's. An associated myocardial insufficiency may cause low voltage of all the leads and

tachycardia. If the septum is involved, incomplete block with prolonged PR and dropped beat, complete block, or bundle branch block may be present.

Serial electrocardiograms may reveal changes indicating activity of myocardial damage. Usually, rheumatic myocarditis heals without any impairment of myocardial conduction and a normal electrocardiogram is found as indication of complete healing. In some cases the scar tissue may interfere permanently with normal conduction. Inverted T's or various forms of heart block remain permanently and unchanged for years as residue of an old healed damage.

## **5 Pericarditis**

The roentgenogram shows the cardiac shadow enlarged in all directions, gradually decreasing in size with absorption of the fluid.

If pericarditis is associated with impaired myocardial conduction the electrocardiogram shows changes, as low voltage or inversion of all T's, which are valuable clues in doubtful or unsuspected cases of pericarditis. In the majority of cases when recovery takes place, T's will be normal again, in others, inversion of all T's may remain permanently as a sign of a scar tissue in myocardium or pericardium, which interferes with a normal myocardial conduction and as such is of no clinical value.

## **6 Bacterial Endocarditis**

The roentgenogram and electrocardiogram reveal changes due to rheumatic heart disease but neither the roentgenogram nor the electrocardiogram are of much value as far as the bacterial endocarditis itself is concerned. If cardiac failure sets in as a complication, it will manifest itself by enlargement of cardiac contour in all directions on the roentgenogram and low voltage of all leads with tachycardia in the electrocardiogram as indication of myocardial insufficiency.

Hemorrhage, embolus, mycotic aneurysm with thrombosis or rupture in the vital organs of the body may be the cause of death in spite of successful treatment of the bacterial endocarditis, and sterilization of the blood stream with penicillin.

# **III ARTERIOSCLEROTIC HEART DISEASE**

Atherosclerotic process involves the aorta, mitral and aortic valves, and coronary arteries and may be associated with abnormal rhythm in the auricles, auricular flutter, and fibrillation.

## **1 Atherosclerosis of the Aorta**

The intima is altered by atheromatous plaques and calcification with increased severity from arch down toward the abdominal aorta.

The weakened wall may dilate, causing a diffuse dilatation and tortuosity of the aorta, more frequently so when associated with hypertension. Clinically, this condition is unnoticed by the patient and does not cause any symptoms, unless rupture of the weakened and dilated aortic wall occurs. It may rupture completely and cause a fatal hemorrhage or may rupture partially with dissection of the coats of the vessel by blood (dissecting aneurysm).

If aorta is dilated and tortuous, the roentgenogram will show bulging of the ascending aorta on the right upper cardiac contour, prominent aortic knob, sometimes with calcification noted on the border, and the wide descending aorta will be seen as a diffuse shadow with a straight border extending from the aortic knob down and to the left of the pulmonary artery. Frequently, the tortuous course of the thoracic aorta may be followed within the cardiac shadow down to the diaphragm. The wide aortic arch and thoracic aorta can be better visualized in the left oblique view.

Lateral view summarizes the wide abdominal aorta, especially when calcification is present.

## **2 Nodular Calcifying Aortic Stenosis**

Aortic stenosis causes a marked hypertrophy of the left ventricle. The electrocardiogram of left ventricular strain and roentgenographic evidence of left ventricular hypertrophy are similar to that described in aortic insufficiency. After reaching a maximum limit of hypertrophy, the left ventricle dilates and fails. Unrelieved, the cardiac failure may be the cause of death.

## **3 Auricular Fibrillation**

As evidence of arteriosclerotic heart disease, the roentgenogram may show left ventricular hypertrophy or enlarged heart in cardiac failure, and the electrocardiogram may reveal other abnormalities besides auricular fibrillation.

## **4 Coronary Sclerosis and Myocardial Infarction**

The coronary sclerosis may be silent and inactive with slight changes in QRS, ST, and T in the electrocardiogram as the only evidence, if the conduction in ventricular myocardium is impaired, or various degree of heart block, incomplete, complete or bundle branch block may be present, if the septum is involved.

The coronary sclerosis may be active. The electrocardiogram, when compared with those taken previously, will show changes in various abnormalities in QRS, ST and T found before.

The myocardial infarction is usually found in two locations (1) in the apex anterior part of the left ventricle and adjoining anterior portion of the interventricular septum, so called anterior infarct with changes in the first and precordial leads, (2) in the base posterior part of the left ventricle and adjoining posterior portion of the interventricular septum, so called posterior infarct, with changes in second and third leads

In myocardial infarct the unaffected portion of the left ventricular myocardium may fail causing myocardial insufficiency with dyspnea, tachycardia, and low voltage of all leads in the electrocardiogram

The myocardial infarct may be the focus for cardiac irritability and cause appearance of premature systoles or paroxysmal auricular tachycardia, flutter or fibrillation

The localized necrosis in the myocardium leads to a localized involvement of endocardium with formation of a mural thrombus and of pericardium with pericarditis. The thrombus may lead to embolus in systemic circulation and if a vital organ as the brain, is involved may be the direct cause of death while the myocardial infarction itself may be clinically improving

The necrotic myocardium may rupture. If the wall ruptures the hemorrhage may cause heart tamponade and death. The rupture of the interventricular septum may heal without any ill effect leaving an opening in the septum

The acute myocardial infarction may heal without leaving any clinical or electrocardiographic evidence of impairment. In others the infarcted area causes localized thinning and bulging of the wall a ventricular aneurysm and impairment in myocardial conduction with permanent changes in QRS ST or T as a residuum of a healed infarct. Ventricular aneurysm with a well functioning myocardium of the unaffected portion of the left ventricle may not cause any symptoms. If the unaffected portion of the left ventricle fails, myocardial insufficiency unrelieved may lead to death

## IV HYPERTENSION

### 1 Systemic Hypertension

Hypertension in systemic circulation of any etiology causes hypertrophy of the left ventricle the same as in aortic insufficiency or stenosis. The electrocardiogram shows left axis deviation or, in marked hypertrophy left ventricular strain. Left ventricular failure may set in at this stage or after reaching a maximum limit of hypertrophy, the left ventricle dilates and fails. Unrelieved, the left ventricular

failure with dyspnea is followed by that of the right, with cyanosis, peripheral edema, ascites, enlarged liver, hydrothorax, and finally death. Hypertension with left ventricular hypertrophy as shown on the roentgenogram and the electrocardiogram is not hypertensive heart disease. It may be called so, if cardiac failure is associated with and caused by hypertension.

## **2 Pulmonary Hypertension (Cor Pulmonale)**

Increased pressure in pulmonary circulation as in pulmonary end arteritis (see Plate 56) causes hypertrophy of the right ventricle, cor pulmonale, and dilatation of the pulmonary artery and its branches. This is well shown on the roentgenogram: bulging of the left cardiac border in the region of the pulmonary artery, and increased hilar shadows due to dilated secondary branches of the pulmonary artery.

The right ventricular hypertrophy will cause right axis deviation and, if marked, right ventricular strain in the electrocardiogram.

The systemic hypertension causes hypertrophy of the left ventricle, while pulmonary hypertension that of the right. The condition can be recognized by changes on the roentgenogram. The electrocardiogram shows, left or right, axis deviation or ventricular strain.

## **V SYPHILITIC HEART DISEASE**

The syphilitic process in the proximal aorta, aortitis, may spread toward the aortic valve, causing aortic insufficiency and left ventricular hypertrophy as shown on the roentgenogram and electrocardiogram. Aortitis may involve the mouths of the coronary arteries, causing coronary disease as shown in the electrocardiogram. The symptoms and electrocardiographic changes are similar to atherosclerosis of coronary vessels.

A syphilitic aortitis may cause a localized dilatation and bulging, saccular aneurysm in the aortic arch which can be well shown on the roentgenogram.

## **VI CONGENITAL ANOMALIES**

### **1 Congenital Dextrocardia**

On the roentgenogram the cardiac apex and aortic knob are seen on the right side, as the heart is a mirror picture of a normally placed heart. The gas in the stomach may be seen also on the right side, as a transposed heart is usually associated with transposition of abdominal organs. The situs inversus involves the bronchi and the lungs. Lipiodol visualizes transposition of the straight right and the slant left bronchus.

The electrocardiogram shows changes due to transposition Leads 1 and 4 inverted, Leads 2 and 3 replace each other

## 2 Coarctation of the Aorta

The dilated intercostal arteries arising from the thoracic aorta may cause erosion of the ribs as shown on the roentgenogram

## 3 Patent Ductus Arteriosus

Due to increased blood volume the pulmonary artery is dilated and prominent on the roentgenogram

## 4 Widely Patent Foramen Ovale Auricular Septum Defect

As the left auricle is situated anatomically above the right the arterial blood of the left auricle in widely patent foramen ovale (see Plate 66) enters the right auricle and mixes with its venous blood. To take care of the extra load the right ventricle and the pulmonary artery with its branches will markedly hypertrophy. The roentgenogram is quite similar to that in cor pulmonale (see Plate 56) with exception of more outstanding findings of right ventricular hypertrophy. In widely patent foramen ovale the right ventricle is exposed first to the increased pressure, while in cor pulmonale the pulmonary vessels are the first affected by increased pressure.

The contour of a normal right ventricle in the anterior view (see Plate 1) is not visible. The large central and anterior portion of the heart is the wall of the right ventricle. The upper portion of it is the pulmonary conus of the right ventricle which lies medially to the pulmonary artery border. The lower portion is the diaphragmatic surface to the left it is adjacent to the left ventricle and to the right is the right auricular appendage.

In widely patent foramen ovale with marked hypertrophy of the right ventricle (see Plate 66) the pulmonary conus is bulging prominently on the left mid cardiac contour. While the bulging may be due to a certain degree to dilated and hypertrophied pulmonary artery, the greater part of it is due to hypertrophy of the pulmonary conus, which overshadows the hypertrophied pulmonary artery. The hypertrophy of the left portion of the right ventricle causes its rounding and enlargement to the left and when marked, overshadowing the normal straight left ventricular border. The hypertrophy of the diaphragmatic surface adds to the rounding and enlargement to the left and also to the right by pushing the border of the right auricular appendage out. Thus the heart is of globular shape with a marked bulging of the pulmonary conus added to it.

The right ventricular hypertrophy causes right axis deviation or right ventricular strain in the electrocardiogram.

## **5 Pulmonary Stenosis With or Without Ventricular Septum Defect**

The congenital pulmonary stenosis may occur at the valve itself or on the tip of the infundibular portion i.e., the pulmonary conus of the right ventricle (see Plate 68)

In pulmonary valve stenosis, the pulmonary conus is markedly hypertrophied and bulges on the mid left cardiac border, similar to that in acquired type (see Plate 18)

In infundibular pulmonary stenosis, the pulmonary conus is the poststenotic portion of the right ventricle and is not visible on the roentgenogram because it is atrophic and hypoplastic

The right ventricular hypertrophy causes right axis deviation or right ventricular strain in the electrocardiogram

## **6 Tetralogy of Fallot**

Tetralogy of Fallot is a congenital anomaly with dextroposition of the aorta, ventricular septum defect, pulmonary stenosis and right ventricular hypertrophy

See description of Plate 69, also note dextroposition of the aorta on the roentgenogram and right axis deviation indicating right ventricular hypertrophy in the electrocardiogram

As only a part of the venous blood of the right ventricle passes through the pulmonary artery into the pulmonary circulation the insufficient oxygenation is compensated by polycythemia with increased number of red cells and increased hemoglobin, thus supplying sufficient oxygen to the tissues, with a result that the amount of oxygen in the blood is nearly normal

## **CLINICAL PATHOLOGY**

The frequently encountered heart conditions can be divided etiologically into five groups: rheumatic, arteriosclerotic, hypertensive, syphilitic, and congenital. Rheumatic and syphilitic are due to an infection, arteriosclerosis is a degenerative process of the arteries while hypertension is an increased resistance in the peripheral blood vessels due to various causative factors some unknown

### **I RHEUMATIC FEVER**

#### **1 Active Stage**

Active rheumatic fever, affecting the heart causes active rheumatic heart disease. Fever, leukocytosis and a high sedimentation rate are some of the general signs of activity while the appearance of a murmur, electrocardiographic changes pericardial rub are some of the local signs of activity in the heart. Active rheumatic fever may

affect the endocardium with special predilection for valvulitis, mitral or aortic insufficiency, or both. The myocardium may be affected primarily or secondarily by extension from the endocardium. The inflammation may involve the ventricular wall or the interventricular septum or both. The electrocardiographic changes, as prolonged PR, dropped beats or complete auriculoventricular block suggest septum involvement. If present, it is a sign, which in correlation with other findings may lead to a correct diagnosis, but the absence of it does not exclude active rheumatic fever. The pericardium, like same as the myocardium may be affected primarily or secondarily from extension from the endo- and myocardium, causing carditis or pancarditis. Pericardial rub, roentgenograms and electrocardiographic changes, as low voltage inversion of all T's, are some of the important diagnostic signs. The electrocardiographic abnormalities if present are valuable clues in doubtful or unsuspected cases of pericarditis.

## 2 Inactive or Healed Stage

The stage of activity and healing is followed by an inactive stage of a healed process with anatomical and physiological adjustments. Following mitral insufficiency, a fibrosis of the valve causes a mitral stenosis which is an indication of a healed condition, with a moderate or marked hypertrophy of the left auricle and right ventricle to compensate it. In aortic insufficiency left ventricular hypertrophy sets in early, as well as physiological adjustment of the blood pressure. In myocarditis and pericarditis electrocardiographic changes may completely disappear or if the scar tissue and fibrosis interfere with conduction permanent electrocardiographic changes may remain as residuum of a healed condition.

At this stage a murmur as evidence of a valvular lesion, auricular or ventricular hypertrophy electrocardiographic and roentgenographic change are signs of an inactive healed and well compensated process, and the patient does not need any treatment for his condition.

## 3 Cardiac Failure, Stage of Breakdown

If for any reason a breakdown of compensation occurs in inactive rheumatic heart disease a cardiac failure with failure of the function of the ventricular myocardium sets in and the failing heart must be helped by the physician to carry on the required physiological processes or a complete breakdown will lead to death.

At this stage of breakdown of compensation the physician must evaluate the extent of anatomical and physiological impairments in the heart, he must correlate the electrocardiographic and roentgenographic findings as for instance, the extent of auricular or ventricular



hypertrophy with clinical signs of left or right ventricular failures. The knowledge of the degree of cardiac damage and its adjustment by the heart is most helpful to the physician as to the proper dose of the medication to be used.

#### 4 Bacterial Endocarditis

Bacterial endocarditis is a complication of inactive rheumatic heart disease. In addition to septic fever, enlarged spleen, positive blood culture, and cardiac murmur, the bacterial endocarditis manifests itself by various vascular phenomena: (1) emboli, as cerebral or Osler's nodes on fingers and toes, (2) arteritis, with thrombosis and mycotic aneurysm, as of cerebral or coronary blood vessels, (3) hemorrhages, as cerebral, renal, or petechial. If these manifestations affect the vital organs, they may cause death in spite of successful treatment of the bacterial endocarditis and sterilization of the blood stream with penicillin. The same is true, when bacterial endocarditis is complicated by cardiac failure.

## II ARTERIOSCLEROSIS

Arteriosclerosis is not a disease but a degenerative process involving the peripheral arteries.

### 1 Atherosclerosis of the Heart and Aorta

Atherosclerosis of the heart and aorta involves the aorta, mitral and aortic valves, coronary arteries, and may be associated with abnormal rhythm in the muscles, auricular flutter, or fibrillation. In the atherosclerosis of the aorta, the intima is altered by atheromatous plaques and calcification, with increased severity from the arch down toward the abdominal aorta. The weakened wall may dilate, causing a diffuse dilatation and tortuosity of the aorta more often so when associated with hypertension. Clinically, this condition is unnoticed by the patient and does not cause any symptoms, unless rupture of the weakened aortic wall occurs. It may rupture completely and cause a fatal hemorrhage or may rupture partially with dissection of the coats of the vessel by blood. In dissecting aneurysm, death may not occur immediately, but may be postponed for days, weeks, or even months.

The atherosclerosis of the aortic valve may cause aortic insufficiency, with a soft diastolic aortic murmur, characteristic blood pressure changes and left ventricular hypertrophy. This condition if followed or complicated by left ventricular myocardial (cardiac) failure may be called then arteriosclerotic heart disease.

Fibrosis and sclerosis may cause slight aortic stenosis, with soft systolic aortic murmur or nodular calcifying aortic stenosis with pro-

nounced blowing systolic aortic murmur, small plateau pulse, normal or slightly lower blood pressure and marked hypertrophy of the left ventricle. Here again, if this condition is followed or complicated by left ventricular myocardial failure, it may be then called arteriosclerotic heart disease.

## 2 Coronary Sclerosis and Myocardial Infarction

Atherosclerosis of the intima of the coronary arteries is a degenerative process similar to that in any other arteries. The coronary sclerosis may be silent and inactive with slight changes in QRS, ST, and T as the only evidence, if conduction in ventricular myocardium is impaired, or various degrees of heart block incomplete, complete, or bundle branch block may be present, if the septum is involved.

Thrombosis and occlusion of the lumen of a branch of a coronary artery with a resulting localized myocardial infarction and necrosis is a complication and medical accident due to arteriosclerosis of coronary arteries. This is most frequently encountered in two locations: (1) in the apex, anterior part of the left ventricle and adjoining portion of the ventricular septum, so called anterior infarct, a part supplied by anterior descending branch of the left coronary artery and (2) in the base, posterior part of the left ventricle and adjoining posterior portion of the ventricular septum so called posterior infarct a part supplied by the circumflex branch of the left coronary, or the right coronary artery. In myocardial infarct, especially when extensive the unaffected portion of the left ventricular myocardium may trail the myocardial insufficiency in this case being the result of a marked coronary insufficiency. Clinically, there will be dyspnea as a sign of left ventricular failure. In some cases the myocardial infarct may be the focus for cardiac irritability and even appearance of premature systoles or paroxysmal auricular fibrillation, flutter, or fibrillation. The localized necrosis in myocardium leads to a localized involvement of endocardium with formation of a mural thrombus and of pericardium with pericarditis. The thrombus may lead to embolism in systemic circulation, and if a vital organ as the brain is involved may be the direct cause of death, while the myocardial infarction itself may be clinically improving. The necrotic myocardium may rupture with clinically improving. The necrosis causes heart tamponade and death. The rupture of the ventricular septum may heal without any ill effect leaving a small opening in the muscular portion of the septum.

The active myocardial infarction may heal without leaving any clinical or electrocardiographic evidence of impairment. In the others the infarcted area causes localized thinning and bulging of the wall.

ventricular aneurysm, and impairment in myocardial conduction with permanent changes in the electrocardiogram. Clinically, there may be dyspnea as a sign of left ventricular failure.

The episode of myocardial infarction may be followed later by another in the same or another area, with consequences as already stated.

### III HYPERTENSION

#### 1 Systemic Hypertension

Increased tension in peripheral arteries is an abnormal finding and not a disease. To overcome the high peripheral resistance, i.e., the higher diastolic pressure, the power of the heart, i.e., systolic pressure increases with resulting hypertrophy of the left ventricle. When dilatation of the left ventricle sets in with myocardial insufficiency, dyspnea and left cardiac failure as physiological and clinical evidence of it, we are dealing then with hypertensive heart disease. If unrelieved, the left ventricular failure with dyspnea will be followed by that of the right with cyanosis, peripheral edema, ascites, enlarged liver, hydrothorax, and finally death.

#### 2 Pulmonary Hypertension

Any obstruction in pulmonary circulation as endarteritis of the pulmonary arteries causes pulmonary hypertension with hypertrophy of the right ventricle, so called cor pulmonale. The right ventricle may fail and cause death.

### IV SYPHILIS

Syphilis of the heart affects the proximal portion of the aorta, the aortic valve and the ostia of the coronary arteries. Clinically, syphilitic aortic insufficiency is similar to that of rheumatic or arteriosclerotic etiology. If the hypertrophied left ventricle fails, the cardiac failure unrelieved, may lead to death. The occlusion of the mouths of the coronary arteries may interfere with normal coronary circulation and cause coronary heart disease. Syphilis and arteriosclerosis of the heart frequently coincide.

A syphilitic aortitis may cause a localized dilatation and bulging, a sacular aneurysm in the aorta. Symptoms when present are due to pressure on or erosion of adjacent structures.

Aortic aneurysm may rupture and cause the patient's death.

## V CONGENITAL ANOMALIES

Congenital anomalies are various defects in the structures of the heart and large blood vessels. When an anomaly causes a cardiac failure it may be called then a congenital heart disease.

Coarctation of the aorta is an example of congenital anomaly without abnormal communication or shunt between the right and left side of the heart. Patent ductus arteriosus, auricular and ventricular septum defects are examples of arteriovenous shunt with arterial blood entering the venous circulation. In tetralogy of Fallot, in addition to pulmonary stenosis there are ventricular septum defect, dextro-position of the aorta and right ventricular hypertrophy. Tetralogy of Fallot is an example of venoarterial shunt, venous blood of the right ventricle mixing with arterial blood of the left causing cyanosis.

This kind of mixture is possible because of dextro-position of the aorta and venous blood of the right ventricle entering the aorta directly while increased right ventricular pressure due to right ventricular hypertrophy permits the venous blood of the right ventricle to enter the left ventricle through the ventricular septum defect. Polycythemia is a compensatory adjustment mechanism for the insufficient oxygenation of the blood.



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